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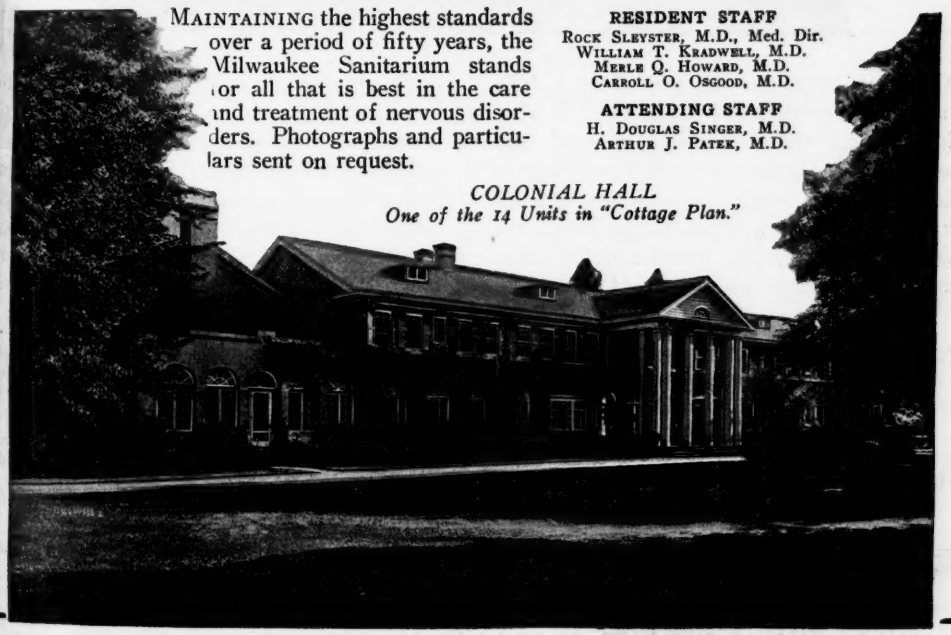

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ILLINOIS MEDICAL JOURNAL

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Editorials

ONE OF THE ADMITTED EVILS OF STATE MEDICINE IS THAT POLITICS WOULD HAVE ITS FINGER ON THE WHEEL

Minimum of statesmanship and maximum of politics are two highlights in the inherencies of state medicine.

A snake and his shadow are far more separable than are state medicine and politics. But in this credulous country of ours it may take a complete wreckage of our entire social structure to convince the man who wants something for nothing, that no such relationship endures in mortal life; that Santa Claus is a phantasy of childhood, and that even if Peter is robbed to pay Paul, that the debt must be met eventually if eternal equilibrium is to be maintained.

That farcical body, with its more farcical report, The Committee on the Costs of Medical Care, has done as much damage to the general public as the child who tossed a match into a gasoline tank, because the day was cold and the villagers were suffering from the weather.

Staunchly the dissenting minority group stands by its statement that

"There is nothing in experience to show that state medicine is a workable scheme or that it would not contain evils of its own which would be worse than those which it is supposed to alleviate. Above all, there is no evidence to prove that it would accomplish what ought to be the first object of this committee, the lessening of the costs of medical care."

One of the admitted evils of state medicine is that politics would have its finger on the wheel. Where in the United States is there any greater evil than the policies of politics? Among its political inherencies are graft, stupidity, incompetence, intimidation and everywhere the suppression of individual enterprise, together with an ever increasing tax-rate. For this socialized regime in medicine can never exist without state and federal financial aid secured from tax levies

to say nothing of direct local taxation for this purpose. Even if medical committees are allowed to direct (?) the work, the string on such direction lies in the clause that such committees "might be elected by popular vote like school-boards or be appointed by municipal or county officials."

Regimenting medical men under state control means but one thing and that is the dragging of the science of medicine into the gutter of political robbery. One of the country's prominent doctors burst frankly into print a month or so ago on the reason why he did not enter into that branch of the profession that appealed to him the most—i. e. institutional practice. Said this man in part,

"I served on the staffs of two state hospitals. One of these was frankly and openly under political control while the other had been divorced supposedly from politics and placed under a civil service system. Yet there was no difference in the matter of the amount of political interference in the two institutions. State institutions are as their name implies, instruments of the state, and are in the hands of politicians. Shameful, but true. And equally appalling is the fact that the public as an entity, knows no gratitude. And so I saw good physicians, who had been in the service so long as 15 or 20 years, discharged without explanation or ceremony because of some slight difference or misunderstanding arising between them and the politically controlling powers."

Now in these institutions of which this physician speaks, the profession sees state medicine in miniature. What would be the conditions in a system when the profession is controlled by a lay-dictating medical super-bureaucracy that would mulct from the taxpayer an even greater levy than that secured now for the school system, or for the army and navy.

Politics has made a mess of public affairs. Politics has smudged the school system. Politics should not contaminate medicine.

Public health and welfare, humanity in distress, cannot tolerate and should not be forced to struggle against the hand of the big political boss behind the hand that wields the scalpel.

Political mismanagement and bureaucracy should be bridled and bitted before it goes another step.

THE GENERAL PUBLIC LONG THE GOAT OF POLITICAL BOSSES IS NOW MADE THE GUINEA PIG FOR LAY EXPERIMENTERS IN MEDICAL ECONOMICS

One of the first essentials in the traditional recipe for hare soup reads,

"First catch your hare."

Current lay programs for compiling a new medical condition for the United States are based upon the initial fundamental of

"First destroy your doctor and annihilate the rational progress of scientific medicine."

Unfortunately the general public, long the goat of political bosses, is now made the guinea pig for these lay experimenters in medical economics.

The anesthetic these dabblers in economics flourish is a pretended alarm over the meager incomes obtainable now by the bulk of physicians. They are going to uplift, are these makers of hare soup without the hare,—uplift the doctor and his rights and necessities be damned.

The scalpel with which these uplifters plan to do their murderous task is our old friend Compulsory Health Insurance. One endowed philanthropy (?) proposes this tax-supported injustice as an improvement in medical practice.

Exactly how sure the politicians are that this socialistic practice can be put into effect is the statement by Harry L. Hopkins, Federal Relief Administrator, that compulsory health insurance could now, by a bold stroke, be put into effect "in the next eighteen months." He calls it "carrying the American people along"—which sounds a bit like taking them for a ride, or the parable, only this time it is the guinea pigs who constitute the public of Gadarene swine, headed for the precipice.

At a joint meeting of the American Academy of Political and Social Science and the College of Physicians of Philadelphia (February 7, 1934) a frank avowal as to the objectives of such organizations as the Twentieth Century Fund, the Milbank Fund and the Rosenwald Fund came from no less an expert in his line than Michael Davis of the Rosenwald Fund.

The Editor of the *Journal of the American Medical Association* says, a large part of Mr.

Davis' activity consists in efforts to attack medical leadership and to undermine organized medicine as it endeavors to function in behalf of the medical profession.

Living under a system characterized by a most vicious distribution of wealth and a grossly unequal ability to pay for essentials, much less luxuries, yet the uplifters wish to socialize the medical profession first of all in order to palliate this state of affairs instead of abating the factors that make for poverty, the cause of so much illness; and by mass-production methods at a time when individualization of the sick human being was never so necessary and so highly developed. Who, under socialization, would spend hours psychoanalyzing a patient?

William Trufant Foster, an economist of Newton, Massachusetts, makes the suggestion that there is great folly in burdening physicians any longer with business affairs which they have notoriously mismanaged, for which they are not trained, in which they are not interested, and which interfere with that single-hearted devotion to patients which is the glory of the profession. Herein the doctor himself is rated as an imbecile.

This editor is inclined to agree with a fraternity brother in the East, Dr. Samuel Horton Brown, Editor of the *Weekly Roster and Digest,* organ of the Philadelphia County Medical Society, who says that these wild men of the sociologic council, seldom in contact with the sick, ask us to listen attentively to them while they give us directions for the care of the sick. These wild men, says Dr. Brown, are temporary workers in the field holding fat jobs which require them to be articulate in the blah which they spatter upon the medical profession, which profession is composed of permanent workers on a very large scale in this field who have done far more than merely prescribe for the sick.

Dr. Brown suggests that the time is rapidly approaching when there will be an unequal distribution of sociologists, and he asks wickedly whether the sociologists will then entertain any proposals from outside for a change in their procedure. In any case, he concludes, the Marx brothers are now putting up a much better show than these sociologists' groups, and it is a relief to watch the one who is a mute.

THE OLD FASHIONED FAMILY PHYSICIAN AN EXCELLENT THING TO FALL BACK UPON

Discovery by the general public that a general practitioner is a pretty wise man and an old fashioned family physician an excellent thing to fall back upon. Also that a great deal of treatment can be had quite competently in the home.

Discovery by the specialist that general practice is a scientific necessity and also an ever present help to all humanity, even including the specialists. Also that the general practitioner has less overhead, and greater contacts.

The general practitioner has on the average really fared so much better economically, during these lean years that more than one specialist or consultant has ceased to sit in his office, doing literally that, and has gone out to take the air and to care for sick persons by making house calls.

Medicine at least has had some of the old order of sanity and science restored by the depression. Which is as it should be, for this is all bound to re-emphasize the importance of clinical training, of late years far too much neglected.

The real restoration of the doctor must begin, however, in the medical school.

As Dean Lewis of Baltimore says ably "he should know how to make a diagnosis; know the natural course of disease and how to observe it and he should know what therapeutic measures should be instituted to meet the indication and when they are to be employed. During the past few years, emphasis has been laid on the laboratories. This was necessary because such rapid strides had been made in biochemistry, biophysics, bacteriology and the histologic examination of tissue. The laboratories have been obtained and now more stress should be laid on the clinic. More clinical material is required, for in modern medical teaching the technic of diagnostic procedures is no longer simply demonstrated; but they are learned by the students and practiced by the student until these procedures can be used independently. In teaching hospitals an endowment should provide the required number of free beds and the patient should be the patient of the student, who under strict supervision can assume charge. *This is not possible*

under any scheme in which the patient pays full or half rates. The medical training which many students now receive makes the doctor dependent on hospitals, laboratories, technicians, nurses, consultants and specialists. These distinctly influence the attitude of students and have a deciding influence in the development of specialism. Importance of physical examinations cannot be overemphasized, for the student of medicine who observes well, percusses well, hears acutely and feels intelligently has advanced far in the way of diagnostic ability. With a due apportioning of diligence, the essentials of anatomy, physiology and pathology can be mastered. During the brief years of pupilage the details of the various branches cannot be grasped so that all cases can be accurately diagnosed and successfully treated. A deep knowledge of pathology is the foundation stone of diagnostic ability. Every effort should be made to bring these practitioners into contact with hospital facilities. Unfortunately, at the present time many of these men from the day they begin practice are excluded from hospital services. The medical profession is largely to blame for the development of specialism and the eclipse of the doctor, for during the past few years the patient has been educated to believe that the specialist is the last word, and as a result patients consult specialists first rather than the doctor. The licensing of specialists or the recognition of certain qualities which specialists should have will limit considerably their number and increase the number of doctors."

Note, if you please, the excellently based protest against the full or part pay clinic.

MISLEADING MATERNAL MORTALITY STATISTICS

For years the ILLINOIS MEDICAL JOURNAL has been pounding away to disintegrate the false premise set before both the profession and the laity through the misleading tables of maternal mortality statistics, that afford such fertile fields for biased and factitious economists.

Disregarding the basic truth that every nation, and in fact, practically every community, has its own classification as to what constitutes "maternal mortality," a group of pseudo-scientists and yearning uplifters attack this question of maternal mortality upon groups of figures lacking entirely a single great common denominator.

Until the same yard-stick is applied to the measurements of all figures, from all nations and from all communities, it is plainly evident that in this question of "maternal mortality statistics" figures can and do lie with the greatest of ease and of inaccuracy.

To evidence this inaccuracy when it comes to international comparison, a very comprehensive citation of findings compiled from statistics issued by the Children's Bureau of the Department of Labor and other sources appeared under date of June, 1934, in *American Medicine,* under the caption, "Maternal Mortality Statistics."

By permission of the Editor of *American Medicine,* this article is herewith reproduced. We quote:

"The general practitioner has been raked over the coals by the report on Maternal Mortality issued by the Section on Gynecology and Obstetrics of the Academy of Medicine. The report ascribed 61% of maternal deaths to the negligence of the attending physician, and has paved the way for numerous unjust criticisms directed against the general practitioner, published in the sensational lay press.

"What does a careful examination of maternal death statistics reveal? It shows, in the first place, that statistics are valueless unless the same standards of tabulation are applied to all countries. In this country the number of maternal deaths is tabulated by the Census Bureau, and the results are published by the Children's Bureau of the Department of Labor. In 1926, they issued a report known as Publication No. 158 and entitled 'Maternal Mortality' by Robert Morse Woodbury, PhD. On page 58, he makes this statement: 'a detailed study of the results of applying the United States instead of the English rules to the deaths in England and Wales in 1920 indicate that the rate in England and Wales would have been increased by about 15% if the United States rules had been applied.'

"In 1920 the deaths incidental to childbirth in the United States were 7.99 per 1,000 women who survived confinements. In England and Wales they were 4.33 per 1,000. Adding 15% to the latter brings it up to 5 per 1,000. This still brings our statistics three per thousand higher than those of a theoretically comparable country. Why the difference? The difference is partly due to our large negro population, who are poor maternal risks owing to contracted pelves from rickets, incidence of venereal disease (very high in the negro race) and poor state of general hygiene. In 1921, the maternal mortality of white women in this country was 6.6 per 1,000 births, and of negroes was 10.8 per 1,000.

"Another reason for the theoretically high rate of maternity mortality in this country is the large number of abortions performed in this country and the fact that our statisticians in Washington draw no distinction between deaths during natural confinements and deaths

arising after abortions. Many women attempt to perform this operation upon themselves. The hazard is so great as to be equivalent to committing suicide. Others have the operation performed by law-breaking physicians or midwives. The risk is still great. Dr. Julius Levy, Director of the Division of Child Hygiene of the Newark Health Department, investigated all maternal deaths in Newark in 1924, 1927 and 1928, and came to the conclusion that in the cases he investigated, abortions caused the maternal deaths in 20% of all cases of maternal deaths and in 60% of those dying within the first six months of pregnancy. These figures are startling and show us the valuelessness of our present methods of recording maternal deaths. As these deaths do not form any part of the normal hazard of giving birth to children and should be tabulated separately. A legitimate practitioner does not perform abortions except in a small minority of cases where it is necessary to save the mother's life, when it becomes legal to do so. An abortion is performed only after a consultation with a reputable physician who concurs in the fact that such an operation is imperative in order to save the mother's life. To saddle the medical profession with the blame for mortality from operations done by the patient herself or the outlaws of the profession is manifestly unjust. Dr. Dorothy Reed Mendenhall in another investigation showed that in 1927, 1,256 maternal deaths occurred in seven states investigated, and 299 of these deaths or approximately 25% were due to abortions. A proper correction of our maternal mortality rate would show that while it is not the lowest in the world, it is less than the average of all countries.

"The lowest rate seems to be in the Scandinavian countries, and because abnormal cases are extremely rare, and the women thus are better risks. A contracted pelvis in those countries is most exceptional. Operations become unnecessary. Pain is less. Shock is less, recovery being uneventful. Any operation has a certain element of risk.

"Pages 25 of the U. S. Department of Labor Bulletin entitled 'Maternal Mortality' admits that no distinction is drawn between deaths from septic full term confinements and deaths from infected abortions.

"The medical profession is striving in every way to reduce the maternal mortality rate to as near zero as possible. This can be brought about only with the cooperation of the patient, classified as good, fair and poor risks. Good risks are normal healthy women and have uncomplicated confinements even if confined by midwives. Most American midwives have a training far inferior to that of European midwives. Especially the negro midwives have no scientific training whatsoever. The untrained midwife is another factor swelling our maternal mortality statistics.

"Fair risks may frequently be converted into good risks by prenatal care. Heart trouble, kidney trouble, diabetes, and venereal disease may all be much mitigated before delivery. These cases are well taken care of by any good general practitioner who has supplemented his medical education by a few months of obstetrical training in a hospital.

"The poor risk should be diagnosed early in pregnancy by the general practitioner, and promptly referred to a specialist if necessary. These cases include marked degrees of contracted pelvis, probably requiring a Cesarean operation (slight degrees of contracted pelvis are very frequent and are properly handled by the general practitionar), toxemias when severe enough to threaten eclampsia, and certain hemorrhagic conditions readily recognized by any good practitioner.

"American methods of gathering statistics are largely to blame for our supposed high rate of maternal mortality. The family physician handles the majority of confinements, and handles them well, and is almost invariably underpaid. In most cases, the physician's fee is well within the people's means, is very moderate, and usually the physician is able to collect only part of his fee, or none of it. Many physicians refuse confinements on account of the smallness of the fee charged by their competitors, in no way commensurate with the work or responsibility involved. A confinement should be considered equivalent in responsibility to an appendix operation. Small fee does not excuse careless work upon the part of the obstetrician, but it might cause some of our better trained men to refuse cases.

"The New York Obstetrical Society disagrees with the report of the New York Academy of Medicine. It would have been wiser for the Academy of Medicine to have made constructive suggestions for lowering the mortality rate, rather than destructive criticism. Why was the report sent to the lay press? The Academy should have issued a booklet to the members of the medical profession, giving recommendations such as:

"1. Proper prenatal care.

"2. Proper aseptic technique.

"3. Avoidance of meddlesome obstetrics. Instrumental deliveries not to be performed unnecessarily.

"4. The dangers in the use of pituitrin.

"It will be noted by the reader that the maternal mortality of New York City, in the report, is 5 to 1,000, while in the country at large it is 7 to 1,000. New York City, in spite of the severe criticism given in the report, compares favorably with most European cities.

"The conscientious physician welcomes constructive criticism so that the maternal death rate would be lowered to an irreducible minimum (a certain number of deaths will always be inevitable because of a definite hazard in bearing children, especially in instrumental cases. The prospective mothers should engage the family physician with confidence, instead of being frightened away from him, to the large medical centers, already monopolizing too much practice. There is no criticism of the manner in which obstetrical cases are conducted in the large hospitals. The aseptic technique and skill are above criticism. However, the family physician is qualified to conduct 90% of obstetrical cases, and can maintain an equally good technique under equally good conditions. If hospital care is preferable to home care, we would suggest that any physician in good standing be given the privilege of conducting obstetrical and medical cases in any hospital. Surgical privileges should also be accorded to those with proper training. The proposition of the closed hospital (found

only in our large cities) is the cruelest injustice ever perpetrated against our long suffering profession. How much longer are we going to permit it?"

EXTENT OF RETENTION OF INGESTED ALUMINUM

According to the experiments of E. W. Schwartze, Gerald J. Cox, Richard B. Unangst, F. J. Murphy and Helen B. Wigman, with the assistance of W. H. Bradley and R. C. Uhlig, Pittsburgh (*Journal A. M. A.*, Nov. 25, 1933), the aluminum content of fresh tissues of guinea-pigs receiving no added aluminum is about 0.4 part per million or less. The carcasses of growing guinea-pigs on a diet containing no added aluminum have a higher content of aluminum than those of the adult animals. The feeding of large amounts of soluble aluminum salts produces a barely detectable deposition of aluminum in the soft tissues (less than 0.5 part per million) and somewhat larger amounts (from 0.5 to 1 part per million) in carcasses. No systemic pharmacologic effects can be ascribed directly to absorbed aluminum. Aluminum does not appear to be cumulative in the tissues. No harmful effects can be expected from soluble aluminum occurring naturally in foods or introduced by utensils into a diet of normal phosphorus content.

BLESSED BE THE OLD-FASHIONED MOTHER!

Thank God, some of us have an old-fashioned mother. Not a woman of the present period, not one that is hand-painted and enameled, with all her society manners and fine dress—whose hands, white and jeweled, never felt the clasp of the dimpled baby fingers, but caresses a bulldog instead. But a dear old-fashioned mother with a sweet voice, eyes into whose clear depth the love light shines, and whose brown hair, just threaded with silver, is lying smooth upon her faded brow. Those dear hands, worn with toil, gently guided our steps in childhood and soothed our cheek in sickness, ever reaching out to us in yearning tenderness and love. Blessed is the memory of the dear, old-fashioned mother. It floats to us like the beautiful perfume of God's grandest blossoms. The music of other voices may become lost and forgotten, but that sublime, radiant and enchanting memory of her will echo in our soul forever. Hurrah for the old-fashioned mother! She doesn't need the help of two brakemen and a conductor to get in or out of the cars. She wears a dress, not an unsightly bag.—*D. A. Anderson.*

PAROXYSMAL TACHYCARDIA

In 3 cases of paroxysmal tachycardia, a high-fat diet, supplemented with acids (and in one case with whiskey), maintained an acid urine and lessened the incidence of attacks of paroxysmal tachycardia.

The acid medication was either 5 grs. of acetylsalicylic acid 4 times a day, or 10 drops of dilute hydrochloric acid before meals, 3 times a day.—Dr. Jas. C. Healy, in *New England J. Med.*, Nov. 19, 1931.

MEDICAL ECONOMICS

FACING THE ISSUE

Some say we live in a great age. Others that we live in a machine age. Still others designate our age as modern and that the old has no place in it; and some think of this age as one of speed. Then there are those who classify this age as one depending on change. That it is a strange and uncertain age most all are agreed.

If one is a bit critical, he could well designate it an age controlled largely by fear, refusing to face the issues of the day and finding much to support his argument. That our age is a combination of all these elements there is no denying. The question remains to be answered, why do we not reduce our wrongs, correct our mistakes and gather unto ourselves the good that advancement in knowledge and skill has produced and made ready for our use?

We do not live in an age of famine but in a harvest of plenty, for never was there a time in the history of the human race when the essentials of life were so plentiful and so easy to produce, and yet we live in want of the good. We live in an age of almost ultra-educational advantages and scientific attainments, yet we are unable to reduce our learning to a common denominator of human benefits. We willfully and knowingly wreck the great things of life and stand crying before the wreckage our own strength and knowledge produced.

We are not satisfied in wasting the good of our own generation but in our egotism we deny the good of the past and draw a curtain of old fogyism between the past and present and expect a great future instead of drawing the straws of good of the past through the present and connecting the future.

The medical profession is only one part of the great program of human endeavor. It has no right to any special place only in so far as it makes a place for itself. This it has done through the efforts and sacrifices of those who have gone before us.

The medical profession has no right or reason to obtain or demand anything for selfish purposes and as far as the public is concerned the profession has met the obligation, but in our relations one to another have we been as unselfish as we should? I fear not and this is the reason for our economic troubles today. We do not need to learn how to win from one another,

to cheat or even compete. What we need to know is how to live with one another in a land of plenty. There is no need of these things. There is, however, a need of closer contact and a willingness to stand by our ethics that the world may realize we are worthy of our hire.

We are complaining more about our economic condition than we are honestly and fairly working to right it. All that is wrong with our economic structure we permitted to be. What we need is the courage to face the issue and make right our wrongs. They are our problems to solve in a medical way and can only be solved by us of the profession. Only physicians can understand medical economics for our economic problems have little in common with business economics. Economics, as they relate themselves to the practice of medicine are not a science but a problem. Like a man's stomach, as long as he is not conscious of having one it is all right. It is only when it hurts that he knows he has a stomach. So it is with medical economics.

We, as a profession, have gone with business and trade through an abnormal period of prosperity. We have enjoyed the luxury that went with it and we have permitted a bit too much commercialism and selfishness to creep into our professional lives. There was a great opportunity during this period to do this unnoticed and we must not be blamed for this too much as it was human, but we can be blamed if we insist on keeping these faults that do not belong to us.

The scenes have shifted. Over-values are gone and the world stands in need. We must readjust our economics to fit the pattern of modern change and make them harmonize with the day in which we live. Our greatest need, as we see it, is the need of the things we formerly had in those days of false prosperity. If we wanted these to continue we should have saved more of the money we made and more wisely invested what we did save. We have placed this false need in front of us and have become victims of fear, due to a dread of what we consider a desperate need. The instinct of self-preservation has returned within us paralyzing our reason and we have permitted conditions to enter our profession that have no place within our ranks.

Let's not lend an ear to the prophets of disaster and despair and cast aside the calm sense of reasoning that has been ours in the past. We must meet the new order with a profession equipped to meet the medical needs of the public and our economics will adjust themselves to this new order if we will only remain true to ourselves.

Organized medicine has the intelligence to meet its economic problems. It only remains to use this intelligence in the right way. It has been said that nine-tenths of wisdom is knowing when to use it. Let's redeem ourselves from selfishness and fear, meet our issues with medical sense, with the full knowledge that the world cannot exist without doctors, that within our profession is the power to lift all undesirable problems without and still strength remains to carry on, without a tinge of the feeling of weakness.

I have no fears of the foes without. My fear is of the foes within. Let no man in the medical profession become so big that he can harm organized medicine and our issues are met face to face and will be solved to the good of all, remembering that medical economics does not have for its base the dollar but patients well served medically and well pleased with their doctor.

CHARLES S. SKAGGS, M. D.
President, Illinois State Medical Society.

INCREASING WEIGHT OF THIN PATIENTS BY INSULIN

Four thin patients gained weight rapidly following the administration of insulin. As a result of the treatment there was a great increase in the appetite.

The treatment is begun by administering 3 units of insulin subcutaneously every 3 hours. The patient should eat liberally ½ hour to ¾ of an hour after the injection to avoid insulin reaction. The dose of insulin is gradually increased until 10 units or more are injected every 3 hours. The insulin is discontinued when normal weight is reached.—Drs. L. H. Nahum and H. E. Himwich, of New Haven, Conn., in *Am. J. Med. Sc.*, May, 1932.

STEINACH THERAPY

From the endocrine point of view, the Steinach operation (vasotomy and ligation) could be called the opposite of castration. . . It adds life to the years, not years to the life.—Dr. Harry Benjamin, in *Am. Med.*, Dec., 1932.

LIVER IN ANEMIA

A case of primary anemia, which failed to respond to both liver extract and ventriculin, responded satisfactorily to the juice of whole liver.—Drs. H. A. Freund and A. E. Price, of Detroit, in *Ann. Intern. Med.*, May, 1932.

Correspondence

BEG YOUR PARDON!

In the issue of the ILLINOIS MEDICAL JOURNAL for October, 1934, appeared without due, accurate, and proper credit to *American Medicine,* reproduction of the bulk and the substance of a statistical article upon "Maternal Mortality Statistics." This patently unintentional offense occurred through an oversight in the proof-reading which failed to correct the omission by typographical error of the credits to *American Medicine* which appeared in the original copy furnished the printer. We apologize.

SOCIALIZATION OF THE PRACTICE OF MEDICINE

During the next session of Congress legislation for the socialization of the practice of medicine through the passage of a health insurance law similar to that now in force in Britain, will come up. A bill is now in the making which will be revolutionary in nature and is expected to be passed as an emergency measure. The member of Congress from your district needs to know your thoughts regarding this legislation and it is your duty to properly inform him NOW. Here is our view in this matter.

What we need in our lives is a creed, not just a code. The health insurance law, as now in force in England makes a sham and mockery of the practice of medicine. It is pauperizing to the public, degrading to the medical profession and extravagant to the nation. It is not practical and though it may be enforced by the authority of the government it is not satisfactory to any one and cannot endure.

It pauperizes the public by encouraging indolence with the most trivial of illnesses and injuries, by contributing to malingering through inviting the individual to magnify his incapacity to work, to feign suffering or deformity, and to conjure up ailments through which he can evade work. Any executive of a penal or charitable institution can testify to the unbelievable lengths to which inmates will go to avoid the usual routine duties of the establishment.

It degrades the physician by tempting him to always diagnose serious ailments which will require prolonged, constant and frequent attentions. He will be more concerned with the number of times he attends his patient than with searching out and removing the causes. He will extend himself socially rather than studiously because "midnight oil" will be unprofitable. The hale fellow will prosper and the thinker will starve.

It will be extravagantly expensive by occupying the doctor's hours with minor ailments which can very well be cared for by domestic remedies, filling his waiting room with semi-social callers who consult the doctor frequently because it doesn't cost anything, and by schemers who will connive with the doctor, or intimidate him, into certifying to a length of illness beyond what actually exists. Lastly, socialization of the practice of medicine will enroll an army of civilians for clerical and statistical work whose regulations and routine delays will seriously handicap emergency medical and surgical work thus jeopardizing the patient's convalescence and at times his life.

National health insurance as now practiced in Britain is the despair of every one and the comfort of none. It is a burlesque on the scientific practice of medicine, a violation of every traditional, ethical and progressive tenet. Were not the results so lamentable, the comedy would be laughable. It is the chagrin of every honest physician who labors to give relief to suffering mankind and a disappointment to those seriously sick who do not appreciate the trickiness of the whole plan. Its adoption would be a definite setback to public health and therefore we view it as a "something for nothing bait." *If you have any stamina tell your Congressman what you think.*

Public Relations Committee.

STATUS OF THE AMERICAN SOCIETY OF TRAUMATIC MEDICINE AND SURGERY

Chicago, Ill., May 8, 1934.

To the Editor: Representatives of the Insurance Medical Directory, Inc., have recently solicited surgeons in and about Chicago to assist in the organization of a Society to be known as the "American Society of Traumatic Medicine and Surgery."

It is proposed that 100 surgeons throughout the country become charter members of this proposed organization. These men would all receive

their membership in the organization free of charge "as and when the same shall be organized," and the remainder of the members were to pay a certain sum per year for their membership, "which said sum shall entitle me to registration in the directory of the Insurance Medical Service, Inc., and to all publications of the *Journal of Traumatic Medicine and Surgery,* without further cost."

The Insurance Medical Directory has already been published with more than 500 names of surgeons included in the directory. Several of the surgeons whose names are included have never authorized the use of their names in that directory. Representatives of the Insurance Medical Directory, Inc., are now soliciting names of other physicians and surgeons to be included in this directory at the rate of $25 the first year and at the rate of $10 per year thereafter.

In the case of some of us whose names appear in this directory, free of charge, we do not feel that the presence of our names therein should be used by salesmen to influence other physicians and surgeons throughout the country to pay out $25 to have their name included in the directory. As one surgeon put it, when he phoned one of the undersigned, inquiring about the directory—"There are some imposing names in that directory, and for that reason I would like to be in it; but I don't want to pay $25 to get in it and $10 a year thereafter to stay in it unless you advise me to do so."

Because of the above facts the undersigned surgeons felt that the medical profession of the State of Illinois should be informed concerning their position with regard to this proposed organization which is as follows:

1. The organization of an Association to be known as the "American Society of Traumatic Medicine and Surgery" is not needed.

2. If a National Traumatic Association is ever to be organized, it is the function of surgeons, and not a lay organization, to develop such an organization.

3. The present plan of the Insurance Medical Directory to organize such a society and to collect membership fees from physicians and surgeons throughout the country is condemned by the undersigned.

4. None of the undersigned have authorized the above corporation to use their names in their campaign to organize the above mentioned society. Furthermore, the undersigned demand that the use of their names in this connection cease at once.

Signed: Philip H. Kreuscher,
Harry E. Mock,
Wm. Cubbins,
Geo. G. Davis,
Leroy P. Kuhn.

ILLINOIS DEPARTMENT RESERVE OFFICERS ASSOCIATION DESIRE CO-OPERATION

Office of Vice-President,
Illinois Department, R. O. A.
Federal Building,
Rock Island, Ill.

October 1, 1934.

To the Editor: As the Vice-President of the Illinois Department Reserve Officers Association, I have been assigned the duty of membership. There are no doubt many medical corps officers in the state of Illinois who have not been properly approached or properly advised as to the function of the Reserve Officers' Association and hence are not in the organization.

The Illinois Department R. O. A., will appreciate any assistance which you may be able to render in the matter of securing all medical corps reserve officers as members of the R. O. A., and I wish to thank you now for your help in making this an accomplished fact not only through your letters to members but through resolution presented to your state society when it regularly convenes.

W. T. Stevenson,
Major, Engr-Res.,
V. P. Ill. Dept. R. O. A.

IN DEFENSE OF THE INSURANCE COMPANIES

September 22, 1934.

To the Editor: In the September issue of the Illinois Medical Journal I noticed a letter by Ray B. Essick (I presume of Illinois) entitled "Another Chiseling Insurance Company."

I grant that a rather ambiguous situation is presented, and yet some reason can be seen for the same from the insurance company's standpoint.

Section 42 (b) of the Illinois Motor Vehicle

Act, by an amendment approved July 2, 1931, provides as follows:

"Provided, however, that no person riding in a motor vehicle as a guest, without payment for such ride, nor his personal representative in the event of the death of such guest, shall have a cause of action for damages against the driver or operator of such motor vehicle or its owner or his employee or agent for injury, death or loss, in case of accident, unless such accident shall have been caused by the wilful and wanton misconduct of the driver or operator of such motor vehicle or its owner or his employee or agent and unless such wilful and wanton misconduct contributed to the injury, death or loss for which the action is brought.

"Nothing contained in paragraph (b) of this Section shall be construed to relieve a motor vehicle carrier of passengers for hire of responsibility for injury or death sustained by any passenger for hire."

I take it from Dr. Essick's letter that the injured persons were occupants of the car driven by the person assuring the doctor that he would be remunerated.

Under the above Section the State law of Illinois provides against liability on the part of the car owner or driver. There is an exception to this general rule which most insurance companies make, in a provision somewhat similar to the following:

"The company does hereby agree to pay irrespective of the limits expressed (elsewhere in the policy) the expenses incurred by the insured in providing such *immediate* surgical relief as *is imperative* at the time of any accident covered by this policy."

This probably is put in as a protection to the insurance company to cover the general case so that for lack of immediate attention a much greater loss might not be suffered. This provision in the contract thus provides for payment when immediate surgical (not medical) attention is necessary, and does not restrict it to those not riding in the insured's car. This causes the ambiguity that if immediate surgical attention is imperative the insurance company will pay for it, even though the injured are occupants of the assured's car, but if not imperative that such surgical attention be immediate—or even if it is better that it be delayed for several days—then the State Law does come into effect that the occupants of a car cannot recover from the driver or owner.

Furthermore, it must be remembered that in a liability insurance policy the contract is to protect the insured, not to provide accident insurance for any person who may be injured by riding in or because of a collision with the car insured.

GLENN A. TREVOR, J. D.

STATE AUXILIARY ORGANIZATION

Mrs. A. B. Middleton,
Chairman State Organization Committee,
Pontiac.

Realizing the importance and the necessity of a united effort upon the part of the medical men as well as the wives of medical men in the State of Illinois to combat the vicious medical legislation that is being proposed and will continue to be proposed, it is absolutely necessary that the largest and most perfect organization possible be effected at an early date by the wives of medical men of this state, in order that we will be in a position to offer valuable help to our husbands in case the occasion should arise.

It is a pleasure to realize the almost unanimous cooperation among the Medical Societies of this State, proven by the replies to contact letters that have been sent from the State Organization office.

Medical Auxiliaries are being organized all over the state under the direction of the various District Councilors. Judging from the present interest that is being taken, by the time we hold the state meeting in Rockford this year a large percentage of the active Medical Societies will have an active Medical Auxiliary.

It is hoped that if there is a Medical Society which has not given their ladies an opportunity to organize that they will do so at their next meeting in order that their wives will be eligible to attend and take part at the next meeting in Rockford.

LEE COUNTY ORGANIZES AN AUXILIARY

Mrs. W. T. Holladay,
Amboy.

The wives of the members of the Lee County Medical Society met with their husbands at dinner in the Colonial House, Grand Detour, on Tuesday evening, October 2.

Following the dinner the ladies adjourned to meet with Mrs. Lucius Cole, State President of the Woman's Auxiliary, and Mrs. J. P. Simonds, Corresponding Secretary. Mrs. Cole presided and the question of organization was discussed pro and con after Mrs. Cole had presented the whys and wherefores of organization.

A vote taken for organization was unanimous. The following officers were elected:

Mrs. W. T. Holladay, Amboy, President; Mrs. W. A. McNichols, Dixon, Vice-president; Mrs. David Murphy, Dixon, Secretary and Treasurer; Mrs. C. A. Robins, Dixon, Historian.

It was voted to have the dues one dollar a year for the present. Following this action, the meeting adjourned. There were eleven ladies present at the organization meeting.

McHENRY COUNTY ORGANIZES AN AUXILIARY

Mrs. F. L. Alford,
Crystal Lake,

September 27, the wives of the Doctors of McHenry County met and organized. Mrs. A. B. Middleton of Pontiac, State Organization Chairman, was our speaker.

The following officers were elected: Mrs. Frank L. Alford, Crystal Lake, President; Mrs. C. F. Baccus, Woodstock, Vice-president; Mrs. J. C. Sculley, Algonquin, Secretary and Treasurer; Mrs. H. J. Schmidt, Harvard, Historian.

EDUCATIONAL COMMITTEE

Report for October, 1934

SPEAKERS' BUREAU:

61—Doctors presented popular health talks before lay groups. These included the lectures at A Century of Progress, two talks before public health chairmen attending a regional conference of the Illinois Federation of Women's Clubs, two high school assemblies, and the usual number of men's and women's clubs and Parent Teacher Associations.

Comments indicate that these programs were satisfactory and interesting: "Splendid talk made and our members were very much interested." "We learned much as the talk was so different from the so called 'common run' of talks."

23—Radio programs given by members of the Chicago Medical Society from stations WGN, WAAF, WJJD.

SCIENTIFIC SERVICE:

24—Speakers scheduled to present scientific papers before medical societies.

COOPERATION WITH COUNTY SOCIETIES:

215—Postal card notices sent to doctors re Perry County Medical Society meeting October 4th.

145—Postal card notices for Medical Woman's Association.

12—Notices to Chairmen of Public Health Illinois Federation of Women's Clubs.

Special publicity Jackson Park Branch public meeting:

750—Cards sent to Parent Teacher Association Presidents and Secretaries.

370—Letters sent to Women's Clubs.

65—Letters sent to ministers enclosing announcement of meeting.

350—Notices sent to south side hotels.

22—Notices to south side libraries.

13—Notices to south side newspapers.

LIBRARIES

180—Press articles on popular health topics to Chicago Branch Libraries.

256—Articles to libraries outside of Chicago.

4—Articles to Red Cross Headquarters in Chicago.

4—Articles to Y. W. C. A.

SERVICE TO WOMAN'S AUXILIARY:

360—Notices of Woman's Auxiliary meeting—November 7th.

275—Letters to Chicago Medical Society Auxiliary members.

50—Notices of Board meeting of Woman's Auxiliary, Illinois State Medical Society.

100—Copies of Agenda of Board meeting October 20th.

325—Revisions for Woman's Auxiliary, Illinois State Medical Society.

NEWSPAPER SERVICE:

20—Articles to newspapers using monthly health column.

446—Regular releases to newspaper using daily or weekly column.

87—Releases to newspapers re Southern Illinois Medical Association.

6—Releases to metropolitan newspapers re Chicago Medical Society meeting, October 24th.

28—Releases Chicago newspapers re public meeting Chicago Medical Society, October 17th.

34—Releases to newspapers re Pike County Medical Society meeting, October 25th.

6—Releases to newspapers re Chicago Medical Society meeting, October 10th.

6—Releases to newspapers re Calumet Branch Chicago Medical Society meeting, October 19th.

2—Notices re North Side Branch meeting of Chicago Medical Society, October 8th.

106—Notices Tri-County Medical Society meeting, October 18th.

Press articles were written and approved on the following topics:

Pulmonary Tuberculosis Is Increasing.
Some Causes of Ear Ache.
Round Shoulders.
Water as a Dietary Factor in Health.
Tuberculosis—A Family Disease.
The Ability to "Take It" a Matter of Health.
Vitamin C Deficiency.

MISCELLANEOUS:

1500—Cards sent to women's clubs and other groups announcing special popular health program sponsored by Chicago Medical Society.

206—Letters sent to Secretaries and Presidents of County Medical Societies enclosing list of suggested topics for medical programs.

25—Package libraries furnished doctors and laymen.

JEAN McARTHUR, Secretary.

LILLY RESEARCH LABORATORIES FORMALLY OPENED

More than a thousand investigators and research workers were present at the formal opening of the new Lilly Research Laboratories at Indianapolis on October 11. The gathering of distinguished visitors representing many noted bodies and famous institutions in this and foreign countries as well, assembled in a mammoth tent erected for the occasion adjacent to the Lilly Laboratories.

At the formal opening exercises, Eli Lilly, head of the Lilly organization, presided as chairman. Mr. J. K. Lilly, chairman of the board of directors, spoke on

"Research in Manufacturing Pharmacy" from 1876 up to the present, when there is so much evidence of the fact that medical science, in becoming an integral part of our social structure, has, in turn, become in a broad measure dependent upon industrial development.

Dr. Irving Langmuir, director of research for the General Electric Company, discussed "The Unpredictable Results of Research." Sir Frederick Banting talked on "The Early History of Insulin." He gave an account of the early experiments conducted by Dr. Best and himself which first demonstrated the existence of Insulin.

Sir Henry Dale, director of the National Institute for Medical Research, London, and secretary of the Royal Society, spoke on the "Ideas in Medicine and Biology."

A GUIDE FOR THE TUBERCULOSIS PATIENT

The Illinois Tuberculosis Association has recently announced the publication of a small booklet entitled, "A Guide for the Tuberculosis Patient." This booklet is available free of charge to physicians in the State of Illinois through the County Tuberculosis Associations affiliated with the Illinois Tuberculosis Association.

The Guide was edited by Dr. Robinson Bosworth of the Rockford Municipal Tuberculosis Sanatorium, and Dr. D. O. N. Lindberg of the Macon County Tuberculosis Sanatorium. It was prepared primarily for the patient who finds it impossible to go to the sanatorium. It gives certain rules to be observed by that patient under the direction of his or her family physician.

According to W. P. Shahan, Executive Secretary of the Illinois Tuberculosis Association, this Guide is not considered to be a substitute for sanatorium care. He says it was prepared to meet a very definite need among the many patients in Illinois who are unable to receive sanatorium care and who must take the cure at home. Mr. Shahan requests that any physician who cannot get a free supply of these booklets from his local County Tuberculosis Association, advise the Illinois Tuberculosis Association, Security Building, Springfield, Illinois, immediately.

THE EXTENT OF SICKNESS

"There are about one hundred million cases of sickness among the people of the United States every year. Some cases are serious, some are trivial. Altogether they cause not only suffering, but also a billion-dollar loss of wages, and require about three and a half billion dollars for the costs of care.

"One hundred and fifty thousand physicians, 70,000 dentists, 200,000 nurses, 7,500 hospitals, 6,000 clinics and 60,000 drug stores are concerned with furnishing medical care and medicines. Five billion dollars are invested in hospitals, clinics, laboratories and in the private offices of physicians and dentists.

"The costs of medical care are of interest to every housewife who is trying to plan her family budget; to every man who is trying to pay his way while he is well and to meet his bills when he is sick; and to all physicians, dentists, nurses, hospitals, social agencies and taxpayers."

MAYO FOUNDATION PROGRAM

A special program of lectures and demonstrations in medicine will be held under the direction of The Mayo Foundation from December 3 to 7, inculsive. Mornings will be devoted to surgery and dry clinics. In the afternoons and evenings medical and surgical subjects, including cardiovascular diseases, diseases of the nervous system artificial fever, roentgen and radium therapy, laryngology, oral and plastic surgery, gynecology, diseases of the endocrine glands and orthopedics, will be discussed.

While this program is arranged primarily for the Fellows of the Mayo Foundation, visiting physicians are invited to attend.

PULMONARY TUBERCULOSIS: RECENT TYPES OF OPERATION

Howard Lilienthal, New York (*Journal A. M. A.*, April 14, 1934), states that the cure of tuberculous cavities with their dangers of locally spreading the infection, of septic absorption or of mixed bacterial contamination and of further destruction of pulmonary tissue is the chief object of operative procedure. He feels that the day must come when a direct biologic attack on the bacillus of tuberculosis will result in the control and, perhaps, in the final disappearance of the disease, but at present one can hope only for an arrest of progress with cicatrization with or without calcification or ossification, which in ordinary circumstances prevents the continued advance of the malady. He divides the methods employed in the treatment of the disease into medical operative and biologic treatment and discusses the four main surgical procedures (extrathoracic procedures, operations on the thoracic wall, transpleural operations not on the lung and attacks on the lung itself), used in pulmonary tuberculosis. He further states that tuberculosis as a disease is not amenable to surgical treatment but that anatomic conditions of a pathologic and threatening nature resulting from the disease may be treated by surgical procedures. The chief object is the obliteration of tuberculous cavities and the conservation of a healthy lung.

COST OF GOVERNMENT

It now costs 14 billion dollars a year to run the various governments in the United States.

In 1931, the gross income of all the farmers of this country—the sums received for all their butter, eggs, wheat, cotton, fruit, etc.—was about 5 billion dollars.

In the same year, the total of all the factory payrolls, large and small, in this country was approximately 5 billion dollars.

It would take all the gross income (not net earnings) of *all* the farmers and *all* the industrial workers in the United States to pay the present tax bills—and there would still be 4 billion dollars left, each year, for our children and grandchildren to pay.—*Committee on American Education.*

Original Articles

A MESSAGE TO THE WOMAN'S AUXILIARY—THE AGE OF NEW THINGS

CHARLES S. SKAGGS, M. D.
President, Illinois State Medical Society
EAST ST. LOUIS, ILL.

We are living in a new age. This expression is but little more than a decade old, yet it has been used so much that it is becoming old-fashioned. If we object to some custom or policy of our time, we are quickly reminded by someone that we are living in a new age. If we speak with regret of the loss of our former culture and respect of the fundamentals of life, we are again reminded that we are living in a new age. We are reminded of this so often that we are beginning to ask ourselves, are we living in a new age and if we are, what really is this new age. What is the difference between this new age and other ages and what will be the history of this age when written?

Every age, new or old, has had its good and its bad, but the age that has produced more good than bad is the age that contributes to the progress of civilization.

There is no doubt but that we have slipped in several of the essentials in the last two decades and this takes in much of this new age. If we enter economics, we find the roads unnamed and unmarked and no one is certain where he is going and no one can give directions to another.

Our social order is none too certain. Industry and labor seem to be in a wilderness of uncertainty. When we hold this new age up in front of us, we find ourselves searching for something that we might want to carry over into the age to come and we are a bit timid about picking out anything. There is a fear that what we select will not endure and stand the wear of time.

Medicine has faced this new age with a firm determination to stand by its traditions and ethics of the past, but the pressure has been so strong that it felt its strength weakening. Realizing this, organized medicine began casting its eyes about for something or someone who would supply the extra strength to hold against the force of this new age.

Some one had said that the doctor's wife was the reserved strength of her tired husband. When the cares of his practice became too great for him to bear, her arms were always ready to take from him a part of the load. Someone began to think about this. If the doctor's wife was so great an aid to him as an individual and the doctor was a part of organized medicine why could not his wife become to organized medicine the same reserved strength that she was to her doctor. This thought glorified itself by bringing into being the Woman's Auxiliary.

It is an old and worthy saying that necessity is the mother of invention. The women's auxiliary is a necessity to organized medicine in these trying days through which we are going, knowing not where. It is one of the products of this new age and as organized medicine looks into this new age to take from it something that would help it hold its place, it selected the Auxiliary as something it believed would endure and stand the wear of time.

Organized medicine has faith in its auxiliary. It has a right to this faith and reasons to expect much from this added strength. It would be a tragedy if the woman's auxiliary failed to meet these expectations for organized medicine has turned over to it certain tasks that are not only important but necessary. If you don't do them they will not be done and if not done, no one can foretell the results and these results are not going to be good, but bad.

This new age has not produced a surplus of good. For this reason we cannot trust to luck to bring things about as we would have them be. Only careful thought that will direct united action can be depended upon. Such a statement may sound and appear over serious. It may be serious but not over serious. I am not one who believes that the woman's auxiliary is just another social order just to meet and seek pleasure. I am not one who would rob you of one moment's pleasure but if you are to have pleasure you must find it in performing the tasks assigned you, for you are in a new age.

If organized medicine fails to hold its lines, the rank and file of physicians are going to be forced into a position that is going to affect each home and in that home is the doctor's wife. Organized medicine believes in the sanctity of the home and the best and highest type of medical service in the home. It believes that each home

is entitled to have its family physician unhampered by politics or socialized medicine in any form or disguise. It believes in the free and independent right of the home to choose its physician and its right to earn enough to meet the medical expense. It believes in the right of the home and the family doctor to adjust its medical costs without the expensive aid of lay organizations and politicians who spend ten dollars of the people's money to save them a dime that they afterwards take from them because they saved it.

The Woman's Auxiliary has the task of carrying this message of organized medicine into every American home. It is not for me to tell you how you can do this, for this is another task that belongs to your organization. But you can't do it by not doing it. Bernard Shaw was asked what he thought hell was and he replied, "Idleness." If he is correct, then work must be heaven. It is going to take work to meet the problems that confront us and the Illinois Medical Society is asking your organization to redouble its efforts. You have done much and each physician in our state appreciates your work, but there is more to be done and none too much time to do it remains.

I would ask each member to let your state president know that you want to put on an extensive program this year and that each individual member and each county unit wants a definite task to do. The strength of your organization will be measured by the individual effort put forth. To do this effectively, you must be interested in your organization, which will give you a desire to be active.

We must regain our lost ground by pushing the personal onward and upward not only to old levels but to new levels. Our bests must be strengthened to new bests to make sure that the profession of medicine shall be what we want it to be.

THE FUNCTION OF THE STATE DEPARTMENT OF PUBLIC HEALTH IN THE CONTROL OF PNEUMOCONIOSIS

FRANK J. JIRKA, M. D.
SPRINGFIELD, ILL.

Pneumoconiosis is defined as lung disease produced by the inhalation of minute particles of solid materials which produce irritation resulting in fibrosis and induration. Inasmuch as the Public Health Department functions largely in a preventive capacity, the term means to us literally a condition of "dusty lungs." When such a process reaches a stage in which organic changes are taking place in the lungs the case becomes a problem for therapeutic management. Unfortunately in many cases, particularly those of silicosis, therapeutic measures are of small value and the ultimate death rate from the occupational hazards is high. It is our purpose, therefore, to cooperate with the Department of Labor in the enforcement of a program that will have as its object the *prevention* of pneumoconiosis or at least the arrest of the process in workers before irreparable physical damage has been done. Our policy concerning industrial hazards and occupational disease, like that in other public health matters, must be governed to promote the welfare of the community in which these problems exist, and ultimately the welfare of the state at large.

The first legislation bearing on the control of occupational hazards was enacted almost forty years ago. At about that time a Bill was passed and approved requiring industrial plants to use blowers upon metal polishing machinery. Other measures were passed from time to time regulating the manufacture of certain products, and relating to labor laws.

Our present program of Industrial Hygiene had its beginning in 1907, by a joint resolution of the Legislature authorizing Governor Deneen to appoint a Commission on Occupational Diseases whose duty it would be to conduct an investigation of the existing industrial hazards and the conditions under which industrial employees were working. The commission in turn appointed a group of investigators who were particularly well qualified for work of this nature. It is of interest to note that this group included physicians (Emery R. Hayhurst and Alice Hamilton) who have since won international recognition for their contributions to the field of Industrial Hygiene. The work of these investigators brought to light a vast number of occupational hazards that had never been effectively dealt with. This investigation and other subsequent studies led to the passage of the Occupational Disease Law which became effective in 1911. Section One of this Act states in substance, *that every employer of labor in this state engaged in carrying on any work or process which subjects*

Read before Section on Medicine at meeting of Illinois State Medical Society, at Springfield, May 15, 1934.

the employees to the danger of illness or disease incident to such work or process, and to which employees are not ordinarily exposed in other lines of work, shall, for the protection of such employees, "adopt and provide reasonable and approved devices, means or methods for the prevention of such industrial or occupational diseases as are incident to such work or process." The next Section makes specific regulations for the control of definite hazards. Quoting, in part, from Section Two of this Act we find the requirement that "in all processes of manufacture or labor referred to in this section which are unnecessarily productive of noxious or poisonous dusts, adequate and approved respirators shall be furnished and maintained by the employer in good condition, and without cost to the employees, and such employees shall use such respirators at all times while engaged in any work necessarily productive of noxious or poisonous dusts."

Since the passage of this law there has been some progress in the control of hazards of this nature.

Early in the movement many manufacturers showed interest in the improvement of working conditions, and when convinced that certain changes would be a mutual advantage to employers and employees, some proceeded of their own volition to make the necessary changes. This tendency has grown as the years have passed, and today, court actions are few. It is a common thing now for manufacturing plants to present their problems to the State Departments concerned, and ask for a survey. They are given the services of persons who are qualified for such investigation.

Pneumoconiosis may be encountered in employees of numerous manufacturing processes. The principal trades in which excessive amounts of dust are produced are: the abrasive industry; all forms of grinding, foundry work, buffing, and sand blasting; the textile industries; in the heavy metal trades irritating and poisonous dusts are raised; also in pottery works and masonry, and in the handling of leather, skins, feathers, wool, cotton, wood, paper, cement, grain and flour. The amount of dust and the size of the particles may vary widely in different industries.

When workers in these trades show signs of disease due to their occupation the reporting of such cases by the plant physician to the state would be a great advantage to both the employer and the workers. It would enable the state to make an investigation with the object of devising methods whereby further disease could be prevented. Section Two of the Occupational Disease Law specifies certain diseases due to the industrial hazards, and the law provides elsewhere that these are compensable and must be reported. The Act also requires the protection of workers in dusty trades by the use of respirators, but it neither specifies the dust diseases as being compensable, nor does it require that they be reported. We are advised by the Attorney General that silica is not provided for in Section Two. (Opinion based on case of Kelley vs St. Louis Smelting Co., Chap. 307 Ill. Sup. Court reports, P. 367).

The reporting of pneumoconiosis (chiefly silicosis) is, therefore, not compulsory under the Occupational Disease Law.

Section Two of the Occupational Disease Act mentions also any processes in which poisonous chemicals, minerals or other substances which are used in harmful quantities, or under harmful conditions. This appears to be clearly expressed, but legal decisions have referred it back to brass, lead, zinc or arsenic and have ruled that this was the intent and purpose of the law-makers. As I have previously stated, Section Two mentions finally the noxious or poisonous dusts, but requires only that industrial plants provide effective respirators for employees who are necessarily exposed to these hazards. Sections Three and Four of the Occupational Disease Act which deal respectively with the monthly examination of employees by a competent physician, and his reporting of certain occupational disease cases, are of great assistance to the state, *but we could accomplish much more if the requirements made by law were broadened sufficiently to include all important industrial hazards, and the diseases for which they are responsible.* The Division of Factory Inspection is charged (Section Eleven) with the enforcement of the Occupational Disease Act, and violations are subject to a prescribed penalty (Section Fourteen). In 1923 an amendment to the Occupational Disease Act was passed, *which implies that disease arising out of and in the course of employment shall be compensable the same as an injury due to accident,* the consideration and handling of the entire matter to come before the Industrial Commission. This made a distinction between common law suits

originating under Section One, and Industrial Board hearings originating under Section Two. This distinction involves chiefly the financial aspects of cases, the common law suits being for sums ranging from ten to fifty thousand dollars, and the Industrial Board awards being based entirely upon proven disability, and the skill and earning capacity of the worker.

We believe that the importance of dusts as industrial hazards, and the necessity that they be specified in the law as such, *have been well shown by the results of certain common law suits in which workers or their estates have recovered damages for diseases caused by these hazards,* and for the prevention of which "reasonable and approved devices, means or methods" had not been provided by employers as required by Section One of the Occupational Disease Law.

As an example we may mention the case of The First National Bank of Ottawa. Administrator, vs. The Wedron Silica Company. (Vol. 35, Ill. Sup. Court Reports, P. 560) in which case damages of ten thousand dollars were awarded by the circuit court of LaSalle county. The worker had developed silicosis and pulmonary tuberculosis with a fatal termination. The decision of the lower court was later affirmed by the Illinois Supreme Court.

The case of Jannusch vs. Webber Brothers Metal Works (Vol. 241 Ill. Ap. Court Rep., P. 1) in which damages of five thousand dollars were awarded is also of interest. In this case the worker's employment had subjected him to the breathing of metallic dusts. He had developed pulmonary tuberculosis which was held to be the result of his occupation, and within the definition of those diseases to which Section One of the Act applies.

We do not feel that these suits should be brought under common law when the Occupational Disease Law so plainly specifies "poisonous chemicals, minerals, or other substances used in harmful quantities or under harmful conditions," and further specifies "noxious or poisonous dusts," both specifications coming under Section Two. We believe that a united effort should be made to clarify the meaning of statements made in that part of the Act. If this proves to be impractical, we would recommend that the entire section be re-written with the object in view of placing pneumoconiosis on the list of reportable occupational diseases and under compensation.

Owing to the risk of pulmonary disease in workers of the dusty trades to which I have previously made reference, it is necessary that effective methods and devices be employed to provide protection against these hazards.

Some years ago a survey of silica grinding plants was conducted by the state and dealt with the size of plants, number of employees, method of manufacture and other details. Both wet and dry methods of manufacture were in use, this being determined by the purpose for which the product was to be used. The point of interest here is that while the wet process is much safer than the dry, industry demands both wet and dry ground silica as necessary to their work. *Technical factors of this nature indicate the advisability of the state including the manufacturer in conferences involving the adoption of new or improved methods,* the practical knowledge of the manufacture being considered essential to a proper solution of such matters.

There seems to be some misunderstanding with regard to the purpose of reporting cases of occupational disease. While the law requires that the industrial physician must immediately report certain cases, we would much prefer that the industrialist feel and know, that by so doing he is not only helping himself but industry in general. Our statistical departments attempt to compute accurate morbidity and mortality incidence. *When the fundamental facts are not reported to us we have no foundation upon which to rest a movement for new and beneficial legislative measures;* and furthermore the attempt to evaluate our preventive efforts is thwarted. The State Departments which are concerned with these matters are greatly handicapped through the failure of industrial physicians to report the full number of cases of occupational disease. We are aware that the plant physician is not entirely to blame for this difficulty. *Some manufacturers are peculiarly averse to supplying this information, utterly failing to realize that the state's sole aim is in the direction of constructive effort in behalf of the manufacturer and his employees.* This lack of cooperation absolutely prevents the accumulation of accurate and valuable statistics which are necessary in our efforts to allay industrial friction and improve industrial health.

In accordance with the plan of the State Department of Health our medical inspector calls on manufacturers and suggests improvements

that are indicated, explaining why they are necessary and what results may be expected. A great many employers are cooperative, and we have found this to be a more satisfactory method of procedure than that of resorting to the more stringent legal measures which, although available if necessary to force the issue, are likely to cause discontent and opposition. Educative talks are made to the worker in order to assist the manufacturer in making needed changes, for the worker is often curiously apprehensive with regard to medical intervention, and fears that any radical changes may eventually result in the loss of his employment. Patience and tact are required, and repeated visits are often necessary before full compliance can be obtained. When good results are once obtained in such instances they are usually lasting.

In conclusion I wish to repeat that the aim of the State Health Department is purely in the direction of cooperation and assistance, together with the education of employees along lines pertaining to their own safety. Our efforts are fruitless without the support of industrial physicians and manufacturers. The actual control of pneumoconiosis will be accomplished only by strong cooperative effort, first, along the line of improved laws; secondly, by exchange of ideas for the framing of such laws; and finally, by united interest in the general employment of recognized preventive measures.

In relation to all phases of this problem the attitude of the State Department of Health is earnestly cooperative and receptive.

LABORATORY METHODS FOR THE DETERMINATION OF ATMOSPHERIC POLLUTION CAUSING PNEUMOCONIOSIS

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The word "pneumoconiosis" has recently been one of the most used and abused words in medical parlance. When Zenker, a German pathologist, invented the term many years ago, little did he realize the difficulties that would arise in its application and interpretation.

The literal meaning of pneumoconiosis is dusted lung; and without further modification or specification of the general or blanket expression, one does not know the kind or amount of dust which has produced it, whether or not there are symptoms accompanying the condition, and whether or not disability has been produced.

There are specific types of pneumoconiosis, such as silicosis and asbestosis (the two characteristic and clinically recognized diseases produced by dust inhalation) in which the findings are well known through international studies and which produce symptoms and disability. Likewise, the etiology of these definitely established lung diseases is well recognized.

Because of these reasons, therefore, the material in this paper will be restricted entirely to a discussion of the laboratory methods for the determination of atmospheric pollution causing silicosis, which is the most frequent specific type of pneumoconiosis encountered in industry.

To those who have had experience with the medical and legal aspects of silicosis, there has been considerable difficulty in establishing etiological relationships with reference to the kind and type of dust exposure; this same situation has obtained in reference to other occupational diseases, chiefly because the physician has had no incentive to adequately study environmental conditions in the various industrial plants, nor has he had the specialized training which is necsary to make such studies, or to appreciate the findings from this type of work. Great difficulties, therefore, have arisen regarding causal relationships and correlating such relationships with the clinical data.

The necessity of the development of objective criteria relating to industrial exposure must be quite obvious to those who have had clinical and legal experience with this problem. This is of especial importance in the differential diagnosis of various chest affections, of which there are many, which so closely simulate silicosis or are imitated by silicosis, in symptomatology and physical findings.

However, before the adequate application of laboratory methods can be accomplished, it is necessary to know the facts concerning the working conditions, which can be secured through what is known as a physical and hygienic survey of the plant, together with an occupational analysis of the men affected. As Bloomfield[1] well says: "The sanitary survey of the workroom

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environment may be likened to the inventory of materials and stock which a business establishment usually undergoes annually. The sanitary survey may well be regarded as a listing of the facilities afforded the workers while in the industrial environment. When one realizes that one-third of the worker's day is spent in this environment, he clearly sees the necessity for a study of all those factors which bear on the health of the industrial workers."

In practice, the physical and hygienic survey consists in carefully filling out an inspection form and recording any additional notes on items which may not be provided in the form. On this form are spaces for data regarding the location of the various types of machines and apparatus used in the manufacturing processes and also spaces provided for a description of the materials used. After securing this information, an analysis of the data is in order and through this analysis one is able to form a picture of the physical and hygienic conditions in each of the workrooms studied and in the plant as a whole.

The occupational analysis permits one to learn of the activities involved and the particular hazards associated with each occupation; such an analysis also discloses the number of persons in each occupation, giving an idea of the importance of each hazard from the point of view of numbers involved. The importance of an occupational analysis to determine the extent of an occupational hazard is at once obvious, but there is still greater value in the subsequent steps necessary for the elimination of the conditions known to be injurious to health. It is impossible to map out a constructive and effective program of prevention or to know what hazards have been having an ill effect upon the health of persons exposed, without definite knowledge of the occupations, and the many processes and activities which may be associated with unhealthful conditions.

Specifically the physical and hygienic survey of the plant together with the occupational analysis provides information on which one can base an estimate of where samples of the air may be taken and how many should be taken in the various locations to secure adequate estimation of the exposure of the employees.

There are four exposure factors, each of which is important, and which should be continuously and consistently related to each other in the evaluation of any dust exposure and also rated in etiological relationships. These four factors are: (a) concentration of dust—this has reference to the amount of dust in the breathing atmosphere and is generally expressed in terms of millions of particles per cubic foot of air; (b) particulate size distribution—this factor is expressed in terms of microns (one micron being one twenty-five thousandth of an inch) this order of size being microscopic in character—we are particularly interested in particles under ten microns in greatest diameter for the reason that it is this size of particulate matter which can enter the alveoli of the lungs; (c) mineralogical composition—this has particular reference to the quartz content of the dust and is of considerable importance because the quartz is the dangerous element in the production of silicosis—this factor is usually expressed as the percentage of quartz contained in the deposited dust; (d) the final factor is the period of occupational exposure and must be taken into consideration with the above mentioned points in assessing the hazard involved or the attempt to discover if silicosis could have been produced under certain environmental conditions. The exposure period naturally varies considerably in each case, but can be of value only when the other elements of exposure are known.

The technique of dust analysis which will be considered in this paper is that which has been worked out during the past twelve years by the United States Public Health Service and adopted as the standard by that organization. Only a brief description of this method will be given, but a detailed account may be secured by referring to the paper of Greenberg and Bloomfield on "The Impinger Dust Sampling Apparatus as Used by the United States Public Health Service."[2]

It should be understood that the sampling and counting procedures are entirely separate and are not done at the same time.

A brief description of the sampling apparatus used by the author is as follows: An electrically-driven apparatus consisting of a small motor properly geared and attached to a pump and blower produces a suction stream which is drawn through an improved and approved type of glass impinger which can be fitted into a calibrated flask. By the use of a rheostat the speed of the motor can be varied so that on the attached

gauge (which has been previously calibrated) the air may be sampled at the rate of one cubic foot per minute, a stop watch being used to register the time. (The reason that the sample is collected at this rate is because the average breathing rate of a man at an average type of labor is one cubic foot per minute).

Another type of collecting apparatus which is in universal use is known as a compressed air-driven suction device which is likewise connected with the impinger unit. This is essentially a Hancock ejector which when connected with the compressed air line in any given plant changes it into a suction stream and registers on a gauge which has previously been calibrated to deliver one cubic foot per minute when the readings lie within certain limits.

After the samples have been collected they are brought into the laboratory and properly diluted either with distilled water or 95 per cent. chemically pure alcohol, whichever was originally used as the collecting medium. There is no formula for the proper dilution, the object, however, being to secure a proper dispersion of the particulate matter so that it will not agglomerate or be superimposed when put under the microscope for enumeration. One learns by experience at about what opacity as gauged by the naked eye, it is wise to make dilutions.

After dilution, the next step is what is known as plating. Approximately one c.c. of the suspended material is pipetted into a previously calibrated Sedgwick Rafter cell of the type which has been used in studying water and sewage samples. The plated sample is then allowed to settle for twenty or thirty minutes, having been covered by a cover slip, and is then ready for counting.

Enumeration is done under a magnification of 100 diameters with an ocular of 7.5 and a tube length which will allow a calibration with a stage micrometer so that the Whipple disc used in the ocular measures exactly one millimeter on each side of the large square. By the use of the mechanical stage on the microscope the Sedgwick Rafter cell is moved about and five enumerations are made in one-fourth of the large ocular square, these five enumerations being made on each of two Sedgwick Rafter platings. The ten enumerations are then averaged and from them is subtracted a similar enumeration which has been done on a control of distilled water or alcohol, so that the amount of dust already in the collecting medium may be subtracted from the average count. Knowing the size of the counting field, the volume of the Sedgwick Rafter cell, the dilution of the sample, the average net count, and the number of cubic feet sampled, one can determine the concentration of the dust in terms of millions of particles per cubic foot of air sampled.

The size of the particulate matter may be noted as one does the enumeration, by comparison with the fine markings on the ocular disc. Another technique for estimating the size frequency of industrial dusts has been worked out by Bloomfield[3] and this is briefly as follows: by the use of an Owens Jet Dust Counter, the atmospheric dust is projected directly on a naked cover slip. The cover slips are mounted on an ordinary microscopic slide and the dust particles are measured by the use of a filar ocular micrometer at a magnification of 1000 diameters, using the oil immersion objective. The horizontal diameter of at least 200 dust particles in several representative fields are measured for each sample. With this magnification it is found possible to measure particles as small as 0.5 micron in size, while particles smaller than this size are easily distinguished at this magnification and their presence recorded.

In assessing the mineralogical composition of any industrial dust, we are chiefly interested as mentioned before, in the percentage of quartz. This procedure is carried out by a petrological examination, and is briefly as follows: a gross sample of deposited dust (not that taken through the impinger collector) is mounted on a glass slide in an oil having a refractive index of 1.54—1.55. The slide is then studied under a petrographic microscope (which makes use of polarized light) with a magnification of 270—360 times, which will permit the determination of the refractive index of the particles examined. All dust particles having a lower index than the oil are considered to be quartz, provided that they do not possess other properties foreign to quartz. All of the grains in the field are counted as well as the quartz grains and the average is thereby derived. One technique is to make a determination in each instance based on 25,000 separate particles. This method has been adapted from the work of Knopf[4] who has described in the

Public Health Reports a method for the quantitative determination of quartz in dusts.

The period of occupational exposure as has been mentioned, is extremely variable in different instances but should be obtained from the employment and medical records and considered in conjunction with the results gained from the evaluation of the other three exposure factors described.

Knowing then, the physical and hygienic conditions, the occupational activities, the concentration of the dust in the working environment, its particulate size distribution, the mineralogical content with reference to the quartz percentage, and the number of years of exposure, one can by comparing with other studies which have been made, assess the severity of the hazard involved and state how it compares with conditions where silicosis has developed.

The criteria which have been suggested by the U. S. Public Health Service relating to environmental exposure in the production of silicosis, were worked out in a series of studies relating to the health of workers in dusty trades and refer especially to exposure to siliceous dust. A description of the material is given in Public Health Bulletin No. 187.[5] It was found for instance, when using the methods previously described that exposure to dust with a quartz content of approximately 35 per cent. and considering particles under ten microns in greatest diameter, when the concentration was less than ten million particles of dust per cubic foot of air the exposure was considered to be relatively safe; however, when exposures to concentrations of more than twenty million particles per cubic foot of air were encountered, silicosis occurred in varying periods of time.

Other studies have been done in relation to other types of dusts in establishing concentration, particle size and the quartz content, so that comparative data may be considered; however, the chief concern today should be regarding the production of the common type of specific pneumoconiosis which produces a definite disease and disability, namely silicosis.

One should never minimize the importance of clinical findings in these studies and it is necessary that the environmental exposure data be thoroughly coordinated and correlated with the clinical information. One cannot, however, obtain an adequate view of the etiological relationships without a quantitative and qualitative estimation of the exposure factors as mentioned.

Something should be said here concerning the technique of the methods which have previously been described. A great many well-meaning persons have literally "taken up" these procedures and without any previous experience of any kind have attempted to make dust determinations. The reputability of these tests, therefore, has suffered severely because of misuse and abuse and the interpretations of the findings have also been poorly done in many instances. Scientific foundation and application of dust determination procedures will never be sound until the persons who undertake the work are given special training and have had sufficient experience to the end that they have developed real expertness.

The real value of the so-called "dust count" has been a source of confusion. A dust count of and by itself is of no more significance in the estimation of a health hazard in a dusty process than is a blood count or urinalysis alone in making a medical diagnosis. True, these methods yield some information. In connection with the four factors which have to be taken into consideration when estimating a relative silicosis hazard in any industrial environment, the so-called "dust count" refers merely to the concentration of the dust and does not give any information relative to the mineralogical composition, nor yield any knowledge concerning the exposure of various individuals. Even when all of these factors are known, they must be properly interpreted and applied to each set of conditions found in any given plant; they must be coupled with a hygienic plant survey and an occupational analysis of all exposed employees, and also an assessment of the efficiency of the dust removal and protective devices used. When this combination is possible, the use of these methods is of considerable value. In short, a dust study and survey of a plant will yield information regarding the severity of the health hazard and the efficiency of control devices. This information is used in two ways: to prove that the health hazard does not exist, or when it does, to indicate measures of control. It is plain, therefore, that there are good reasons for abolishing the term "dust count" and substituting in its place the expression "dust analyses," or "dust study," or "dust survey."

It should furthermore be well understood that

the technique of dust sampling and dust analysis has been recorded in the literature, and that there is also an approved technique sponsored by the U. S. Public Health Service. It is well that this is so, for we need a national standard, so that comparisons may be made by various investigators, and so that there may be also a basis for comparisons between different plants. But the technique cannot be properly learned by merely reading it and "trying it out;" as previously mentioned adequate training and experience are both necessary to secure real results.

The methods herein described (as well as other analytical methods applying to the estimation of other injurious substances in the industrial environment) may at the present time be applied in three practical ways: 1. The employer may desire to know just what the relative severity of the dust exposure may be in his plant, either for purposes of checking up on the dust control system which he has, for the prevention of future cases of silicosis, or for the defense of claims; 2. The attorney representing an industrial organization may likewise wish to get information concerning the severity of exposure, which information may also be of the same use to the insurance carrier; 3. The casualty and indemnity insurance company before undertaking the underwriting of a risk involving occupational disease may want to know the nature of the exposure, which would permit of more intelligent underwriting and a better way of evaluating the risk. The last application mentioned has been one which has been very much neglected, principally because these methods have not been at the general disposal of insurance carriers and consequently their information for the evaluation of possible future risks has been largely gained through mere visual inspection without the making of any analyses.

Finally, in simple terms, one must know where he has been before he is able to know where he should go—it is highly desirable that scientific procedures should supplant inference and surmise, when considering projects which involve large amounts of money and time, to say nothing of health and efficiency.

SUMMARY

A brief description of the laboratory methods used in the determination of atmospheric pollution causing silicosis, the most frequent specific type of pneumoconiosis, has been given.

To secure the real value in assessing the relative health hazard in any given industrial environment these laboratory methods must be used in connection with the information gained through a physical and hygienic survey of the working conditions, together with occupational analyses of employees thus exposed. When adequate information concerning environmental conditions has been gained by use of the above mentioned procedures, comparisons may then be made with the criteria which have been suggested by official groups, thereby providing a basis for an estimation of the hazards involved in any given problem.

There are ample reasons for believing that laboratory and survey methods can be only properly used and interpreted by those who have had sufficient special training and experience to permit of real expertness.

Similar study and survey procedures will be used more widely regarding various occupational disease exposures in evaluating the essential hazard, the prevention of disease, and the rating of the efficiency of control devices.

330 So. Wells. St.

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THE HEALTH OF WORKERS IN DUSTY TRADES

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The subject of the health of workers in dusty trades has of late been receiving considerable attention from those interested in the various phases of this problem. When one realizes that the workmen employed in the dusty trades com-

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prise one of the largest groups exposed to any one industrial hazard, it is quite apparent that the importance of this problem has not been overestimated. It is by now fairly well established that exposure to certain kinds of dust has increased the mortality rate from respiratory diseases.

Realizing the importance of this problem, the United States Public Health Service in cooperation with the Bureau of Mines began its first study of the health of workers in dusty trades in 1914, in the mines at Joplin, Missouri.[1] Later, in 1923, the Public Health Service inaugurated a series of dust studies under the general direction of Assistant Surgeon General L. R. Thompson. These studies were all conducted in the same manner in order to permit as detailed a comparison as possible between the different investigations. Briefly, these methods of study may be divided into six parts, as follows: 1. examination to determine the general physical condition of the workers under observation; 2. special physical examination to determine the prevalence of specific diseases of the respiratory system and the lung pathology resulting from exposure to the particular dust hazard; 3. record of the nature and severity of the disabling illnesses; 4. analysis and detailed study of the occupational environment; 5. Occupational mortality statistics relating to the specific dust; and lastly 6. autopsies. In brief, the chief value of each of these studies lies in the fact that it represents careful and detailed observation on a fairly large group of persons whose working environment was accurately determined, especially with reference to the nature and quantity of the dust exposure.

EFFECTS OF INDUSTRIAL DUSTS ON HEALTH

Public Health Service Studies. The first six investigations carried out by the Service dealt with the health of workers exposed to dusts in a cement plant, granite cutting industry, anthracite and bituminous coal mining, silverware polishing plant, cotton cloth manufacturing and in municipal street sweeping.[2, 3, 4] Later, briefer studies were conducted in certain slate, granite, marble and talc quarries, while more recently a rather extensive study of the health of anthracite coal miners was completed.

The first six dust studies conducted by the Public Health Service showed that the workers in the granite cutting plants who were exposed to the highest dust concentrations were found to suffer from an excess of pulmonary tuberculosis after 15 years or more of exposure and developed silicosis from 2 to 10 years. In the group exposed to a lesser amount of dust silicosis developed only after prolonged exposure with no excess of tuberculosis. It was found that a close relationship existed between the degree of dust exposure and the extent and severity of the pulmonary damage. Anthracite coal miners were found to have dyspnea, other signs of pneumoconiosis and respiratory sickness; in addition, an excessive mortality from influenza-pneumonia and possibly tuberculosis. Some of the bituminous coal miners had a generalized fibrosis of the lungs and an excess mortality from influenza and pneumonia. Some cement workers showed signs of slight pneumoconiosis with an excess of diseases of the upper respiratory tract. Workers in the cotton cloth and silverware manufacturing plants and municipal street sweepers were negative for respiratory conditions and other illnesses when compared with the general industrial population.

Rock Drilling. For five years, the United States Bureau of Mines studied lead and zinc miners in the Tri-State District, Picher, Oklahoma. The gangue of the ore is chiefly chert, a form of free silica. The results of the physical examination of the workers for the first year of the study have recently been issued by Sayers, Meriwether and Lanza.[5] Of the 7,722 men examined, a group of 1,647 (21.3 per cent.) was definitely diagnosed as having silicosis, not including those diagnosed as having tuberculosis. All the men diagnosed as having first-stage silicosis had worked an average of 13 years, but those men starting in the mines after 40 years of age worked an average of only 8 years.

The work of rock drilling in subway and tunnel construction in New York City is in some respects similar to that of some of the activities in the hard rock mines, at least in so far as the manner in which dust is evolved, the type of dust and the resultant hazard. Recently the result of a study of rock drillers, blasters and excavators was reported by the New York Tuberculosis and Health Association.[6] The diagnosis of the condition of silicosis was based mainly upon roentgenographic appearance. An examination of 208 rock drillers, blasters and excavators showed that 118, or 57 per cent. of the men, had silicosis in various stages of progression. The exposure of most of these men (75 per cent) had been for 20 years or more. The exact inci-

dence and progress of tuberculosis among these workers was not definitely established by this study.

Sandblasting. No study of the health of sandblasters has been reported in the United States. However, a study of the dust exposure of sandblasters was recently completed by the United States Public Health Service in cooperation with the National Safety Council.[7] This investigation indicated, that judged by the nature of the dust and degree of the exposure, a real hazard exists in this occupation. This study also brought out the fact that it is possible to conduct sandblasting with safety, provided properly designed and well maintained equipment is used.

Diatomaceous Silica Workers. Most of the data on the subject of silicosis have been gathered from studies made both in industry and experimentally on dusts containing free silica in the form of quartz. Such studies have been made in the granite industry, pottery trades, hard rock drilling, etc. There are free silica minerals in existence that are not crystalline but of an amorphous form. In California, about 350 miles south of San Francisco, in Santa Barbara County, there exists what is said to be the largest and purest diatomaceous silica deposit in the world.[8] This diatomaceous substance consisting of about 85 per cent. free silica of an amorphous nature is used in various industries. It is an excellent non-conductor of heat, and bricks made from this substance are now used in building furnaces. It is used as a filtering agent because of its lightness, porosity and clarifying qualities. It is also used in the abrasive industry and in making concrete.

Because of the fact that the mining and manufacture of this substance is a new enterprise it has as yet not been possible to perfect machinery free from the dissemination of dust in the atmosphere, and for this reason the workers are exposed to considerable quantities of this dust. Recently an examination was made of 108 workers selected on the following basis: those employed less than 1 year; 1 to 2 years; 2 to 5 years, and more than 5 years. All of the workers were Mexican laborers and the examinations consisted of x-ray determinations, social and medical histories, and clinical examinations of the chests. Dust determinations were also made but these were not reported. Eighty-four per cent. of the workers were in excellent physical condition. Seventy-three per cent. of workers had "clubbed" fingers in various degrees of development. An analysis of the data obtained from this survey show that silicosis among these workers is a slow process advancing with the years of employment. It was found that 60 men showed signs of early pneumoconiosis; 15 were in the moderately advanced stage and 6 in the advanced stage. This latter finding was among workers with a history of exposure of more than 5 years.

Dust Hazard in Abrasive Industry. Artificial abrasives are widely used in industry and for this reason the effect of the inhalation of dusts produced in the manufacture or preparation of these products is of great importance. In 1925, 1929, and again in 1931, Dr. Clark of the Norton Company at Worcester, Mass., reported the results of his study of the workers exposed to aluminium oxide and silicon carbide dust produced in the manufacture of grinding wheels.[9, 10, 11] This investigation showed that during a period of 13 years there were proportionately twice as many cases of pulmonary tuberculosis among those working in the abrasive dust departments as among those in the departments where no abrasive dust occurred. Unfortunately no dust determinations were made in the present study, so that it is impossible to evaluate the dust hazard on the basis of degree of exposure. This study does point out, however, that it is inadvisable for persons who have had pulmonary tuberculosis to work in atmospheres where large amounts of artificial abrasive dusts, of the type present in the plant under consideration, are present. With reference to the kind and severity of lung fibrosis produced by the inhalation of the abrasive dusts present in this plant, Clark's earlier studies showed that of 79 men exposed to the dust for 10 years, roentgenographic examination revealed 12 men with no evidence of silicosis; 43 showed first stage changes and only 3 exhibited second stage or true silicosis. Of these 3, only one had been exposed to the abrasive dust alone, this exposure lasting about 26 years.

In 1931,[12] Kessler of New Jersey, reported the occurrence of over 100 cases of alleged silicosis in the abrasive powder industry, in which the dust consisted of 99 per cent free silica. Of 40 x-rays of cases reviewed by Kessler only 4 could be called ante-primary. The most important finding of these cases has been the allegedly short duration of exposure—varying from 4 months

to 1½ years. The reports are that many of the men have first or second stage silicosis.

Exposure to Silicate Dust. For many years it has been the belief that the inhalation of dusts composed of silicates (combined silica), in comparison with silica (free silica in the form of quartz), was innocuous. Asbestosis, a fibrosis of the lungs caused by the inhalation of asbestos dust, has been described both in its clinical and roentgenographic manifestations by investigators in other countries, but to date no studies have been reported in the United States. Cement dust, which contains about 22 per cent. combined silica, has been studied, and the effects of the inhalation of this dust on workers was discussed in an earlier portion of this review. Dreessen, of the United States Public Health Service, reported the results of a study conducted among tremolite talc and slate workers.[13] The slate dust to which these workers were exposed consisted mainly of silicates, there being less than 3 per cent. of quartz in the dust. The talc was a hydrous calcium-magnesium silicate, being 45 per cent. talc and 45 per cent. tremolite, with no quartz present. Dust determinations were made, showing the intensity of the exposure, and the results of the study seemed to indicate a relationship between the amount of dust inhaled and the effect of this dust on the lungs of workers. Clinical and roentgenographic studies of the workers showed that the silicate dusts of tremolite talc and slate induce a fine, diffuse, bilateral fibrosis of the lungs which is definitely demonstrable in the x-ray. However, in spite of the heavy dust exposure (1440 for talc and 710 million particles per cubic foot for slate workers) it could not be demonstrated that the resultant pneumoconiosis had led to disability even after 16 years of exposure.

Statistical Studies. Recently, Lanza and Vane[14] estimated that there were some 500,000 workers engaged in the manufacturing industries in this country in which an exposure to silica dust was entailed. Their analysis of mortality experience of twelve life insurance companies for the period of 1915-1926 showed that the actual mortality from respiratory tuberculosis among the silica exposed persons was about three times that of a non-silica dust group If one considers their data for certain occupations in metal mines and granite and sandstone quarries, occupations known to be associated with high concentrations of dusts, then the comparison is still more striking, since the actual deaths among those workers was found to be ten times that of the expected deaths. More detailed statistics on mortality from respiratory tuberculosis are afforded by the last report of the Registrar-General of England and Wales (1921-1923), summarized by Britten.[15] These statistics on occupational mortality show the high rate of 1,886 per 100,000 for tin and copper miners while the rate for "all occupied and retired males" was only 150. Some of the other occupations exposed to silica dust presented a similar, although not as great, reflection of the silicosis hazard in industry.

METHOD INVOLVED IN ATMOSPHERIC INDUSTRIAL DUST DETERMINATIONS

From the evidence just presented concerning the injurious effects produced by the inhalation of certain dusts, it is apparent that a knowledge of the properties of a given dust which determine its capacity to produce pulmonary pathology is essential. Numerous investigations of the industrial dust problem indicate that these properties are the chemical and mineralogical composition of the dust, its concentration in the industrial atmosphere, and its particle size.

In order to obtain a sufficiently large sample for mineralogical analysis, it has been found that dust settled out at the worker's breathing level gives a fair representation of the dust present in the air. Such samples are best analyzed by a competent geologist, who is equipped to make use of a combined chemical and petrographic examination of the dust.[16] Since no two dusts offer the same problem it is difficult to lay down general rules for such an analysis. Each sample must undergo a careful examination under the petrographic microscope, and a complete chemical analysis. Only by such analysis have we found it possible to determine accurately the percentage of quartz present in quartz-containing dusts.

It is necessary to know something of the size of the particles in the dust under consideration in order to ascertain whether or not the dust is of a size capable of gaining access to the lungs. Samples of industrial dust may be obtained with the Owens Jet Dust Counter,[17] in which the dust is projected on a microscope cover-slip. The dust on the cover slip may then be measured with the aid of a calibrated filar ocular micrometer at a

magnification of 1,000 diameters. At this magnification it is possible to distinguish particles as small as 0.2 microns in size.

Recent studies[18] of the size of industrial dusts made by one of the authors showed that nearly all industrial dusts suspended in the atmosphere are less than 5 microns in size and that the majority of the dust particles (71 per cent.) was found to be between one and three microns in average diameter. It is obvious that practically all atmospheric industrial dusts are of a potentially dangerous size.

With reference to the quantity of dust present in the air of a workroom it is apparent that when the dust concentration is high the exposed person will inhale a greater quantity in a given period of time than he will when the dust concentration of the atmosphere is relatively low. Since the development of the fibrosis is partially dependent upon the quantity of dust inhaled, this latter item plays an important role in predicting the relative danger of different environments.

The Impinger apparatus is extensively used, both in this country and abroad, for the sampling of aerial dusts.[19] The atmospheric dust is entrained in a suitable liquid medium, such as distilled water and the dust particles in the sample counted with a microscope.

Prior to obtaining dust samples the investigator should become thoroughly familiar with the industry being studied. This is best accomplished by conducting a sanitary survey and occupational study of each workroom.[20] The sanitary survey of workrooms in any plant yields definite information concerning the presence and extent of potential health hazards and often serves as a guide in establishing which hazards require further study in the form of actual quantitative analyses. In other words, the sanitary survey may well be regarded as a listing of the facilities afforded the workers while in the industrial environment. The occupational study permits one to learn of the activities involved and the particular hazards associated with each occupation. Such an analysis also shows the number of persons in each occupation, which gives an idea of the importance of each hazard from the viewpoint of the numbers involved. In the case of a dusty industry, the occupational study shows the dusty occupations, the number of persons in each occupation, the various activities and the time devoted to each activity of any one particular occupation. Experience has shown that only by a systematic and careful study can dust determinations be of any real value in the control of the industrial dust hazard

THE CONTROL OF INDUSTRIAL DUST

In an earlier portion of this discussion evidence was presented which indicated that a close relation existed between morbidity and mortality rates from various respiratory diseases and the exposure to certain dusts in industry. In the study of the dust hazard in the granite cutting industry,[3] it was definitely proved that there is a direct relation between the magnitude of the dust exposure and sickness and death resulting from tuberculosis. It is quite evident, as a result of these studies, that the remedy of the dust evil lies in the effective removal or suppression of the dust to a concentration considered safe. However, no set rules may be established for the mechanical protection to be instituted in an attempt to control industrial dust. Specific conditions encountered in a plant will determine the type of protection to be employed.

The protection of workers against certain dusts known to be toxic may at times be accomplished by the substitution of a non-toxic material for the toxic one. One example of such a procedure is the possible use of a metallic or other type of artificial abrasive for sand in the sandblasting process in those operations in which it is not essential to use sand, a substance high in quartz content.[7] The mechanical enclosure of the dust-creating process also serves to protect the worker. An excellent illustration of this type of protection is afforded by the modern sandblast barrel used in the cleaning of small objects. Sometimes it is possible to protect workers by the substitution of wet for dry processes. In one instance, in the granite cutting study previously mentioned, an operator using a diamond point tool worked the stone wet; the resulting dust amounted to 22 million particles per cubic foot. The same operator was then requested to work the stone dry; as a result, the amount of dust reached 45 million particles per cubic foot. In the weaving of asbestos cloth it has been possible to reduce the amount of dust in the air by wet weaving to one-fourth of the amount present when the process is conducted by dry methods. Another example of this procedure in allaying dust is the use of water in Leyner and jack-hammer drilling in

hard rock although dust traps have been suggested for use in drilling.[21, 22, 23]

One such trap consists of a metal hood or cup designed to enclose the drill steel at the rock surface. No attempt is made to secure a mechanical seal with the rock or between the trap and the drill steel. The dust is removed by an air stream and is carried into an exhaust system connected to the trap. Escape of dust particles through openings between the uneven rock surfaces and the trap is prevented by the air seal produced by the inflowing air. Tests made on this trap under actual working conditions show that the dust concentration at the worker's breathing zone may be kept down to less than 10 million particles per cubic foot with a 60 cubic foot per minute rate of air flow through the hood. When using this trap or hood underground, for horizontal and down drilling a rate of 200 cubic feet per minute through the hood was found necessary to reduce the dust concentration at the breathing zone to less than 10 million particles per cubic foot.

In certain cases, such as in the sandblasting of large castings in sandblast rooms, the practical safeguard to the worker is to provide him with a mask or helmet of the positive type. In the sandblast investigation previously mentioned it was found that when from 5 to 6 cubic feet of practically dust-free air per minute are supplied to the helmet, the worker receives ideal protection. However, one must always bear in mind that the ultimate criterion of protection should be the dust determination of the air within the helmet during blasting and not the quantity of the air supply itself.

In most dusty processes, however, the most effective means of dust elimination are by the use of properly designed exhaust ventilation systems. Since in many instances it is difficult, costly, and at times unnecessary procedure to remove all the dust in the vicinity of a worker, we need first to determine the minimum amount of a certain dust which the worker can apparently tolerate with impunity. Such information can be made available by the type of studies carried out by the Public Health Service (mention of which was made earlier in this paper). For example, in the granite study already referred to it was shown that the threshold dose was about 10 million particles per cubic foot. Studies of dust removal devices used in granite cutting plants at the time this work was conducted showed that a linear velocity of 1500 feet per minute, as measured by an anemometer at the face of the dust removal hood, would keep the dust at the breathing level of the worker to an amount less than 10 million particles per cubic foot.[24]

Later, the workers at the Harvard School of Public Health reported a study of dust control systems for stone cutting.[25] As a result of this study it has been possible to specify the correct design of local exhaust hoods and the minimum air flow requirements for removal of dust generated by the use of pneumatic cutting tools in granite plants. One of the outstanding contributions of these studies was the work of Dalla-Valle,[26] who pointed out the importance of the velocity characteristics in the design of local exhaust hoods in general. This latter work is of fundamental importance in the design of efficient exhaust hoods for the control of dusts and points out the fact that it is the velocity characteristics of a hood and not the static suction, as considered heretofore, that should form the basis of hood design. What is greatly needed at present, now that some of the basic principles have been established, are studies, both experimental and in industry, to establish the minimum air requirements necessary to remove various dusts to a safe level. This type of work should go hand in hand with broad dust studies aiming at the determination of the threshold doses for various industrial dusts.

It has already been shown that in the use of positive pressure respiratory devices in sandblasting, it is important to have a well-designed device supplied with about 6 cubic feet of dust-free air per minute to give full protection to the worker. With reference to respirators of the filter type, there is still much to be accomplished in the design of an efficient and comfortable mask for continuous use. Of great need at present are standard methods for the testing of the efficiency of respiratory protective devices. Such methods have been recently suggested by Drinker,[27] who advocates testing of a device against the particular dust which the respirator is to encounter; these tests to be made both in the laboratory and under actual industrial conditions. It is further suggested that some impartial agency should conduct such tests and certify the devices for industrial use, the work being done on a cost basis.

At this time it is desired to stress the importance of good housekeeping in the effort to combat the dust hazard. Good housekeeping should not only imply the removal of accumulated dust about a workroom but should also embrace sufficient maintenance of dust removal devices and respiratory protective equipment. Systematic inspection of all dust suppression equipment should be an axiomatic principle in every dusty industry rather than the casual attention which it now receives in many instances.

SUMMARY

In this discussion there has been presented a brief review of some of the studies conducted during the past few years on the health of workers in certain dusty trades. These studies have been of material aid in the efforts to combat the dust hazard. However, much work still remains to be accomplished, there being many industrial dusts about which we need more information. Some of these dusts which are now coming into prominent industrial use are tripoli, a free silica composed of quartz and amorphous silica, vitreous or fused quartz, and the dusts arising in the foundry and ceramic industries. Pure talc and its impure variety soapstone, should also be investigated. Such investigations should be carried out both in the laboratory on animals and in industry, where one is in a position to study the effects of the inhalation of these dusts on workers who have been exposed for a long period of years. In industry, all phases of the problem should be studied, including careful occupational histories, clinical and roentgenographic examinations, nature and concentration of the occupational dust exposure and wherever possible autopsy material should be collected. It is only by such complete dust studies that one may ever hope to amass sufficient basic evidence which could then be used in those further steps, namely, the eradication of the industrial dust evil.

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THE CLINICAL DIAGNOSIS OF SILICOSIS

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As most of you know, within the past few years silicosis has become an acute and important problem. It has existed since men first started chipping at stone and has grown constantly with the development of civilization and industry, with the mining, the quarrying, the grinding, the polishing, the glazing and all the other types of work in which silica dust is produced. Men have been disabled by silicosis and have died of it, and of the tuberculosis which it invites, for centuries, but it is only within the past one hundred years or less that the condition has been recognized and studied by the medical profession, and only within the past few years that in this country the problem has become truly acute. It has become so because certain lawyers have found it to their advantage to hunt up men who have been exposed to silica and urge them to start suit against their employers. Before this the employers were either ignorant or not particularly concerned. Now, with hundreds of suits pending against them, they are frantically interested. Much good will come of it, for the exposure of men to silica dust will be curtailed. In the meantime there exists a bad situation. Most of the men for whom the lawyers are filing the suits are tried before juries. The jurymen, ignorant of the whole situation, prejudiced in favor of the individual against the corporation, and influenced by medical testimony which is divergent, find usually for the plaintiff. The companies are being unjustly penalized and the legal and medical professions are being put in an unfavorable light. And because the lawyers take at least half of the money awarded, the men truly injured are not receiving justice.

The clinical findings of any disease should and must be considered in relation to its pathology. In this condition the primary change is a slowly progressing interstitial peribronchial fibrosis, which involves and eventually blocks the lymphatics and the reticulo-endothelial system. The fibrosis as it progresses limits the expansibility of the lung and may eventually reduce seriously the vital capacity. It may also distort and dilate the bronchi and produce compensatory emphysema of the parenchyma, two conditions which favor the development of chronic bronchitis. In most cases its most serious results arise from the injury to the normal defense mechanism of the lung and the consequent predisposition to infection by the tubercle bacillus. The fibrosis and emphysema also certainly increase the resistance in the pulmonary circuit, in some cases to such an extent that the right heart dilates and fails.

There are no symptoms or physical findings which are characteristic of silicosis. The diagnosis depends upon three things: 1. A history of exposure. 2. A characteristic roentgenogram, and 3. The absence of other conditions which can simulate it.

While a man is working in dust the mechanical irritation of the large particles may produce a cough. This is not a manifestation of the disease. Silicosis may progress well into the second stage without producing appreciable symptoms. The only symptom of the uncomplicated disease is shortness of breath; and pulmonary damage must be extensive before this becomes manifest on moderate exertion. By the time dyspnea has become apparent chronic bronchitis will have supervened with occasional attacks of dry pleurisy. The symptoms of morning cough and pleuritic pains will have been added. Hemoptysis, fever, sweats, loss of weight and other evidences of systemic infection are not symptoms of uncomplicated silicosis. Their presence is practically always indicative of the existence of a complicating tuberculosis.

The only constant physical findings are a limited chest expansion and a reduced vital capacity. In its first and often in the second stage these are slight and there are no other detectable abnormalities. Compensatory emphysema may give hyperresonance and decreased breath sounds and chronic bronchitis scattered bronchial rales. In the third stage large areas of fibrosis may produce dullness and bronchial breathing. Constant post ptussal rales in the apices suggest tuberculosis. Clubbed fingers may or may not be present. Cyanosis supervenes only in extreme cases and then usually as a result of failure of the right heart.

The Complications. Chronic bronchitis, emphysema, bronchiectasis, dry pleurisy and failure of the right heart have already been mentioned as

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complications. Spontaneous pneumothorax and pleurisy with effusion occasionally occur, the former either from the rupture of an emphysematous bleb or extension of a tuberculous focus; the latter always as an early manifestation of tuberculosis. I shall speak later of the relationship between silicosis and tuberculosis.

From the symptoms and physical findings one cannot make a diagnosis. All of these may be absent in the presence of well advanced disease and when present are similar to those produced by many other bronchial, pulmonary and cardiac disorders. Emphysema, chronic passive congestion, bronchiectasis, pulmonary arteriosclerosis and certain types of chronic tuberculosis are a few of the conditions which can closely simulate it.

I have said that three things are essential to the diagnosis. The history of adequate exposure is the first of these. To obtain this it is necessary to investigate the individual's various occupations with great care and to trace his employment from the time he first started work. Following a relatively short exposure there may be a free interval of as much as twenty-five years before the appearance of the first symptoms or the onset of the complicating tuberculosis.

Silicon dioxide and magnesium silicate (asbestos) are the only dusts which have been shown definitely to produce disabling fibrosis and to predispose to tuberculosis. In the present discussion I have not time to list the various occupations in which these dusts are encountered. They are very numerous and occasionally very unexpected. Silicosis has been found in a professional tap dancer who scattered sand on the floor before each performance and in farmers and ranchers in sandy districts. It is important to know that wet grinding and drilling are a little more dangerous than dry.

The length of exposure necessary to produce the disease is not definitely known. I say this because practically all of the studies on this subject have been made on men still working at a dusty trade. That they do not develop it for two, three, ten or twenty years does not mean that this length of exposure is required. We know that the fibrosis progresses after the cessation of exposure; we know that there is often a long free interval between exposure and the appearance of symptoms. There is reason to suspect that a man who develops it after ten years steady exposure would have developed it at the same time had he left the work after a year or two.

That aspect of the subject needs further investigation. In general one can say that the length of exposure necessary to produce silicosis depends upon the concentration of the dust and upon some undetermined individual idiosyncracy. Dr. Sappington has already told you of the methods of counting the dust particles and of the standards of safety which have been set by the public health services of the various countries. Within the past few years cases have been reported of death from uncomplicated silicosis less than one year after the beginning of exposure among workers in the scouring powder industry. Sand blasting produces a high concentration of dust as does sandstone grinding and drilling. Men working at these occupations develop the disease rapidly and have a short expectancy. In most industries such as quarrying, mining and foundry work, the dust is less concentrated and the required period of exposure longer, averaging about ten years with a minimum of two and maximum of twenty-five to thirty. No two individuals working under the same conditions will develop it at the same time. Mouth breathing, intercurrent infections and variations in the function of the reticuloendothelial system have been cited as the explanations of this variation in susceptibility.

A characteristic x-ray is the second essential to the diagnosis of silicosis. The earliest changes of first stage silicosis have been described as an increase of the hilus shadows and of the linear peribronchial markings extending into the lung fields. I myself have never been able to recognize this stage. Every x-ray of the chest has hilus shadows and linear peribronchial markings and in normal individuals both of these vary markedly and proverbially, both actually and with the hardness of the film. First stage silicosis on the x-ray is an exaggeration of normal markings and in my experience it has been extremely difficult to say where the normal ends and the pathological begins.

Second stage silicosis is a very definite roentgenographic entity. It may be either infiltrative or nodular, either a definite increase in the linear markings or distinct peribronchial nodules. The middle lung fields are those earliest and chiefly involved, the right usually earlier and more extensively than the left. The apices and bases are ordinarily spared until late and are

practically always less densely infiltrated than the middle fields.

In third stage silicosis there develop larger areas of opacity and in extreme cases a whole lobe or lobes may be densely fibrotic. It has been stated that cavities develop, but I will have to see these and determine by post mortem examination that tuberculosis is not present before I will be convinced that simple silicosis can produce them.

The Differential Diagnosis. There are numerous other diseases which can closely simulate the x-ray findings of silicosis. The most common are tuberculosis and the chronic passive congestion secondary to heart disease. Rarer ones are miliary carcinosis, pulmonary arteriosclerosis and miliary infection with yeast and fungi. The history and the physical and laboratory examinations are chiefly valuable in identifying or ruling out these simulating conditions. I shall speak later of the differentiation from tuberculosis. When chronic passive congestion is the cause there are practically always physical, x-ray and electrocardiographic signs of heart disease. If heart disease is present, one must be sure that it is primary and not secondary to silicosis. Myocardial failure or hypertension shows on the electrocardiogram a left ventricular preponderance while in that secondary to pulmonary fibrosis the right ventricle is chiefly affected. Mitral stenosis, which also shows a right ventricular preponderance, may be recognized by the thrill and murmurs. In pulmonary arteriosclerosis the red blood cells are usually increased in number and the cyanosis is out of proportion to the x-ray findings. The mycotic infections are febrile diseases with characteristic organisms in the sputum.

SILICOSIS AND TUBERCULOSIS

Silicosis is the only pulmonary condition which is known definitely to predispose to tuberculosis. The incidence of phthisis among workers in silica dust is many times that in the general population. In a majority of cases silicosis gives no symptoms until it is complicated by tuberculosis and most of those who die of silicosis die of tuberculosis. Tuberculo-silicosis progresses rapidly and is usually fatal.

It is often very difficult to tell whether or not the infection has been implanted on the original condition. The x-ray findings of second and especially of third stage silicosis closely resemble those of simple tuberculosis or of tuberculosis and silicosis. The findings suggesting tuberculosis are pleurisy with effusion, constant post-ptussal rales in the apices, a preponderance of shadows in the apices, cavity formation, and fever, and loss of weight. A definite diagnosis can be made only by finding tubercle bacilli in the sputum.

It is often very difficult to distinguish simple tuberculosis from silicosis and tuberculosis. The bronchiogenic spread that occurs after hemorrhage produces x-ray findings that are practically indistinguishable from second stage silicosis. Widely disseminated fibroid tuberculosis, is, as far as I am concerned, indistinguishable from the third stage of the dusty lung. One can say that silicosis is not present only if portions of the middle lung fields are free of all disease. The similarity between the x-ray pictures of silicosis and miliary tuberculosis is well known.

An important problem is presented by those patients who, having been exposed to silica dust, develop tuberculosis without showing clinical or x-ray evidence of silicosis. Does the inhalation of the dust predispose to tuberculosis before fibrosis is demonstrable by the x-ray? In other words is there a pre-x-ray stage of silicosis and does this stage invite tuberculosis? It is my belief that there is and that it does, and that it must be assumed that anyone who, having been exposed to dangerous concentration of free S O, later develops tuberculosis, has developed it because of that exposure. That statement will meet with serious criticisms and strong objections, but I believe that it is true.

There is another important and unsolved problem. What is the x-ray appearance of the lungs during the long free intervals which so frequently occur? Can one say that if, when a man stops work, he shows no x-ray evidences of silicosis, he is safe and will not later develop the disease?

SUMMARY

1. The clinical diagnosis of silicosis is often easy, often difficult.

2. There are no symptoms or physical findings which are diagnostic.

3. Dyspnea is the only constant symptom and limited chest expansion the only constant physical finding.

4. The clinical diagnosis rests upon three things, i. e.,

   a. A history of exposure.

b. A characteristic roentgenogram, and

c. The absence of conditions which can simulate it.

5. The complications of silcosis are pulmonary tuberculosis, chronic bronchitis, bronchiectasis, emphysema, dry pleurisy and spontaneous pneumothorax.

6. Silicosis must be differentiated clinically and roentgenologically from pulmonary tuberculosis, miliary tuberculosis, chronic passive congestion, miliary carcinosis, pulmonary arteriosclerosis, miliary infections with certain yeasts and fungi, and a rare type of generalized bronchiectasis.

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THE PATHOLOGY OF PNEUMOCONIOSIS

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Introduction. In his classical book on occupational diseases published in 1717, Bernardo Ramazzini referred in detail to pulmonary disorders that occurred in people employed in dusty occupations. Little attention was given to Ramazzini's work and many years had to pass before the diseases caused by dust were made the subject of systematic studies.

The pneumoconioses (pneumon-lung, konis-dust) may be caused by organic dust or anorganic dust, the latter being of much greater importance. Of the anorganic pneumoconioses the following types are usually distinguished: the anthracosis due to coal dust, the chalicosis due to dust rich in calcium salts, the silicosis due to free silica and the siderosis due to iron. There are two forms of siderosis of the lungs, namely the red iron lung which occurs in people exposed to ferric oxide dust and the black iron lung that is due to the inhalation of ferrous oxide. In recent years it has become recognized that it is chiefly the dust rich in finest silicon (quartz) particles which is dangerous while most of the silicates are relatively harmless. Coal dust and metallic dust seem to be of little significance if not combined with free silica. Thus, pneumoconiosis now is often identified with silicosis. Recently the fine fibres of asbestos (magnesium, calcium silicate) have been found to cause a severe pulmonary fibrosis (asbestosis) which takes a more rapid course than silicosis. My discussion will be restricted to the two most important forms of pneumoconiosis: silicosis and asbestosis.

Pathogenesis. The amount of dust that is retained by the lungs depends upon the concentration of the dust particles in the inhaled air, the duration of the inhalation of the dust containing air and the size of the dust particles. The lighter the dust and the finer the dust particles the more dust is deposited in the lungs. They are only the particles of less than 0.01 mm. diameter which enter the alveoli. The larger particles settle upon the mucosa of the upper air passages from where they are eliminated by the action of the epithelial cilia, the contraction of the bronchial musculature and the secretion of the mucus glands. The irritation of the upper air passages by the coarser dust particles accounts for the fact that most of the cases of pneumoconiosis show a bronchitic component. In silicosis, this bronchitic component is slight, probably because of the small size of the quartz particles.

The majority of the silicon particles which enter the alveoli are less than 0.003 mm. in diameter and many of them may be so small that they are below microscopic visibility (Policard). Those which can be seen under the microscope appear as tiny, irregular crystals with sharp angles. These crystals are colorless and double refracting. According to Jones the quartz crystals are of less significance than minute fibres of hydrated silicates of aluminum and potassium (sericite—a mineral belonging to the mica group). Jones found the same minute fibres in the rocks which caused severe silicosis. Whether Jones' conception is correct remains to be shown.

The action of the inhaled silicon particles upon the lung may be twofold, namely mechanical and chemical. The mechanical action is suggested by the sharp angles and edges of the minute crystals. Behr and others attribute the greatest significance to the traumatisation of the tissue. Silicosis is, however, an extremely slowly progressing disease and, once deposited, the silicon crystals continue to exert their deleterious influence notwithstanding the discontinuation of the exposure. It has been shown that finely distributed silicon is gradually dissolved in Ringer solution and Policard and with him the majority of the modern investigators assume that in the

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tissues silicon is slowly transformed into a colloidal solution. This transformation is favored by the weakly alkaline reaction of the tissue fluids and the enormous free surface of the billions of tiny crystals. Policard calculated that the free surface of the silicon particles in the lung of a miner equals to almost half a square meter. Colloidal silicon is very toxic (Gye and Purdy) and like other electronegative colloids is stored by the reticulo-endothelial cells (Kettle). In discussing the relations between tissue fluids and silicon crystals Scheid speaks of "surface action" which comprises an intricate combination of processes among which absorption, electric charge, surface tension, hydrophilia and hydrophobia and giving off of molecules are the most important ones.

The concentration of the dust particles is greatest in the portions of the lungs with the most active respiration (Tendeloo). In these portions, namely the lower part of the upper lobe and the upper part of the lower lobe (the middle lung field), the circulation of the lymph fluid is most active which secures the quick resorption of the alveolar content. Hence, in the apical region and in the basal portions of the lungs the silicatic changes are but slight (Flemming-Moeller, Bergstrand).

In general, silicosis develops very slowly and it takes at least from 10 to 12 years before the changes are so marked that they cause clinical manifestations. Stewart points out that the most severe changes are found after an exposure of from 32 to 42 years. There are, however, cases of what is called "acute silicosis" (MacDonald, Piggot and Gilder, Gerlach and Gander, Chapman, Gardner, Kessler, Giese, and others). In these cases manifestations appear already after exposure of from four months to two years. Several factors seem to favor the acute development of pulmonary silicosis, the most important one being apparently the excessive amount of very fine dust with long daily exposure. Some of the cases of acute silicosis occurred in plants which manufacture scouring powder. It has been suggested, therefore, that a high content of the air of free alkali may favor silicosis. The studies of Nicol on workers in fluor spar mines are very interesting. The dust in the fluor spar mines contains only a small amount of free silica and yet the miners develop silicosis rather rapidly with distinct x-ray findings after three years exposure. Nicol suggests a combined action of the fluor and silicon.

It is not the purpose of this brief review to discuss the chemical findings in silicosis. Suffice it to say that the opinions differ as to the silicon content of normal lungs. Some investigators consider a silicon content of the dried lung tissue of more than one per cent. as indicating silicosis. According to McNally the silicon dioxide content of the normal dried lung tissue averages 1.13 mg. per 1 gr. Any lung containing over 2 mg. of silicon dioxide per gram dried tissue indicates undue exposure to a dusty atmosphere. In this connection I would like to emphasize that the decision whether silicosis is present or not must rest with the pathologist and not with the chemist since the anatomical changes of silicosis are well characterized and since only the anatomical changes determine disability and death. There are several factors at work in bringing about the dangerous pulmonary fibrosis some of which are not chemical as shown by the fact that in different mines with identical silicon content of the air the incidence and severity of the pulmonary silicosis varies considerably. Much additional work is required in order to explain the differences which quite often exist between the chemical silicon content of the lung and the extent of the anatomical lesions.

Of the people working under apparently identical conditions in the same mine, quarry, tunnel or factory only a certain percentage develops silicosis. Great differences also seem to exist with regard to the rate of progression and severity of the pulmonary process. In order to account for these differences reference has been made to differences in the mode of breathing, to an individual disposition to fibrotic response (keloid disposition of Schridde) and to the importance of complicating infections. None of these theories has proved fully satisfactory and I believe that a close scrutiny of the mode of exposure of each case may be more helpful than the search for individual factors.

The asbestos fibres which enter the lungs in cases of asbestosis are considerably longer than 0.01 mm. One has questioned, therefore, whether these fibres reach the pulmonary alveoli. According to Gardner the asbestos fibres remain in the respiratory bronchioli from where they are carried into the stroma by phagocytic cells. Stewart makes the statement that, on an aver-

age, asbestosis is fatal after from 13 to 15 years. The pulmonary lesions are more diffuse than in silicosis with a distinct predilection for the basal portions. Asbestos is found in the lungs in two different forms, namely as black and opaque particles and as pecular bodies (asbestosis bodies—Cooke, McDonald, Lynch and Smith, et al.). These asbestosis bodies are pathognomonic of asbestosis and can be easily detected in sections and scrapings of the lungs and in the sputum, especially after enrichment with antiformin or digestion with sodium hydroxide. The bodies are up to 0.075 mm. long and are composed of small discs with rounded margins arranged in form of a tapering rod with bulbous extremity (Hadfield and Garrod). They are golden yellow or brown in color and give a distinct iron reaction. In lung sections they are often seen surrounded by foreign body giant cells. The asbestosis bodies result from changes which the asbestos fibres undergo in the tissues. The soluble parts of the fibres are removed by the tissue fluids and a delicate frame of silicon remains which absorbs proteins. Thus, the needle shaped frame becomes surrounded by a mantle of proteins which coagulate and into which the silica is dispersed. This initiates the disintegration of the frame which is finally resorbed (Berger).

Gross Pathology. In the early stages of silicosis the findings are not characteristic. There is a slight to moderate increase of the connective tissue about the larger bronchi and blood vessels which accounts for the accentuation of the hilus markings on x-ray examination. After an exposure of from one to two years the first characteristic nodules appear. They are very small, from 0.3 to 0.4 mm. in diameter and are rather felt than seen. The nodules gradually increase in number and size, and after about five years measure from 0.7 to 0.8 mm. in diameter (Gardner). Nodules larger than 1.5 mm. are rare (Mavrogordato, Gardner, Giese). They are spherical in shape, sharply defined and very firm, protruding as dark gray or black granules over surfaces made by cutting. They can be readily seen on the pleural surface of the lungs. The dark coloration is due to coal dust, the purely silicotic nodules being pearly gray. The nodules are most numerous in the midportions of the lungs and are first seen along the bronchi and blood vessels and on the pleural surface. Later, the nodules are found throughout the lung parenchyma. The appearance of the nodules is associated with a moderate alveolar emphysema. In the early stages the silicotic nodules are difficult to distinguish from healing or healed miliary tubercles (Tesseraux and Ruiz).

In the later stages, the nodules coalesce. Indurative, sclerosing changes about the nodules which are usually attributed to complicating infections cause the lung tissue to become gradually replaced by a very dense and deeply anthracotic scar tissue. The intensive antracosis is the result of the blocking of the lymph vessels by the silicotic process (Cummins). Solid, deep black and firm areas are finally found which occupy the midportions of the lung sparing the apical parts and the base which become emphysematous. This emphysema may be so marked that by rupture of one of the blebs a spontaneous pneumothorax develops (Behr). At this stage the lungs are usually firmly adherent to the wall of the chest.

In the center of the diffusely consolidated areas which cut like wood one finds often small cavities which are surrounded by friable tissue and are filled with a thick fluid black like India ink. These cavities are from one-half to one and one-half centimeter in diameter. The liquefaction of the dense scar tissue has attracted much attention (phthisis atra) and I fully agree with those investigators who stress that the cavities are due to a non-infectious, autolytic liquefaction of the poorly vascularized scar tissue (Giese, Gerlach, Koopman, Stern, Staehelin, Badham and Taylor). In addition to liquefaction the scar tissue may undergo calcification and eventually also ossification (Giese).

The replacement of large parts of the lungs by a very poorly vascularized scar tissue and obliterating changes in the branches of the pulmonary artery interfere greatly with the pulmonary circulation. The increase in resistance to the blood flow causes hypertrophy of the right ventricle of the heart, the wall of which becomes thickened to from 6 to 10 mm. as compared with a normal thickness of from 3 to 4 mm. With the exhaustion of the reserve power the hypertrophic ventricle becomes decompensated and failure of the right heart is a common cause of death in advanced pulmonary silicosis.

The silicotic induration of the lungs is usually associated with similar changes in the tracheobronchial lymph nodes. There are cases in which

the silicotic induration is more marked in the lymph nodes than in the lungs. The purely silicotic lymph nodes are moderately enlarged. They retain their shape and do not become adherent to adjacent structures. They are deep gray or black in color and may undergo the regressive changes described in the lungs. Perforation of indurated lymph nodes into bronchi or blood vessels is due to complicating tuberculosis (Giese). In acute silicosis the lymph nodes may be spared (Gardner).

The occurrence of silicotic nodules in the liver and the spleen and bone marrow indicates that silicon particles enter the blood stream. In the spleen the nodules may be visible to the naked eye (Giese). Chemical examinations have shown a marked increase in the silicon content of liver, spleen and bone in cases of silicosis. The kidneys contain but little silicon (Hackmann).

In asbestosis, the changes affect mainly the lower lobes. There is a diffuse induration of the lungs with emphysema along the margins, pleural thickening and, in contrast to silicosis, a tendency to bronchiectasis. Gardner says that in asbestosis the fibrosis involves every portion of the pulmonary frame work, alveolar walls, pleura, interlobular septa and the large bronchial and vascular trunks. The changes in the tracheobronchial lymph nodes are insignificant (Gardner). At autopsy the diagnosis of asbestosis can be readily made by demonstrating the characteristic bodies in the fluid expressed from the indurated tissue (Stewart).

Microscopic Pathology. Particulate matter which enters the pulmonary alveoli is quickly taken up by phagocytic cells which are derived from the cells of the alveolar septa (septal cells, alveolar phagocytes). In silicosis too, phagocytosis of the tiny crystals is the first defense reaction and the smaller the crystals the quicker is also their phagocytosis. The crystals injure the cells which swell up, lose their motility and disintegrate. Together with the cellular debris the liberated crystals are taken up by new phagocytes and are gradually transferred from the lumen into the wall of the alveoli and the radicles of the pulmonary lymph vessels. Some of the large, silicon filled cells accumulate in the finest bronchioli and are expectorated or remain in the bronchioli stimulating reactive changes of the bronchiolar walls. Because of the paralyzing effect of the crystals upon the phagocytes (Policard) the transfer progresses very slowly. In the lymph vessels the swollen cells filled with silicon particles and free clumps of silicon crystals pile up and block the lymph flow. The lymph stasis stimulates connective tissue proliferation about the bronchi and blood vessels. From the blocked lymph vessels some of the silicon particles are removed by the local histocytes and carried into the surrounding connective tissue. The majority of the crystals are filtered out in the small lymph nodules along the bronchi and underneath the pleura. Progressing blocking of the lymph vessels prevents the removal of subsequently inhaled dust which now remains in the alveolar wall piling up to larger aggregates.

Wherever the silicon crystals pile up they become surrounded by spindle shaped cells. These cells enter the clumps of crystals and produce collagenous ground substance. Layer after layer of concentrical fibrils is laid down about and between the crystals, the fibrils fuse to coarser trabeculae which become hyalinized and a sclerosed granuloma results which is very characteristic of silicosis. The silicotic granuloma consists of a center of whorls of hyalinized connective tissue with scanty and deeply stained nuclei which is surrounded by a more cellular tissue. Wedged in between the hyalinized trabeculae one finds the fine crystals of silicon and a varying amount of coal dust. The smallest coal particles (soot) are attached to the silicon crystals and Policard speaks of a tattooing of the crystals. The periphery of the nodules contains much more silicon than the center (Belt). The young nodules are arranged about small arteries and arterioles. Policard describes a juxta arteriolar, Giese and Schridde emphasize a periarteriolar location. The inclosed vessels become later obliterated by proliferation of the intima (Gerstel, Giese, Behr, et al.) and only a few elastic fibrils remain. Mavrogordato mentions recanalization of the obliterated arterioles.

In ordinary sections mounted with balsam the silicon crystals escape detection because of their high refraction. In order to demonstrate the crystals in sections several methods have been recommended. Watkins-Pitchford and Moir digested the sections with concentrated nitric acid. Giese mounted his sections in media of lower refraction index (cedar oil, glycerol) while Policard and Scheid made use of the microincineration method. According to my experience the

microincineration gives the most satisfactory results. Examining an incinerated section under the polarizing microscope one sees the silicon particles as brilliant crystals against a velvet black or light gray background. The crystals vary from 0.001 to 0.008 mm. in diameter. Microincineration causes the coal pigment to disappear while the iron granules are easily identified by their rust brown color.

The first silicotic nodules to become visible correspond to hyalinized peribronchial and subpleural lymph nodules (blockade nodules of Mavrogordato). In the later stages the nodules do not show definite relations to preexistent structures. The nodules increase in size by apposition of indurated lung tissue (Bergstrand). Older nodules become often calcified and I believe that the calcium is derived from the tissue fluids and is not related to calcium salts in the dust.

In advanced pulmonary silicosis the nodules form groups which are embedded with a very dense and deeply anthracotic scar tissue. The nodules now are less sharply demarkated than in the earlier stages of the disease. In the fibrosed areas the lung structure is completely obscured and the larger bronchi and blood vessels only can be differentiated. The diffuse fibrosis results from a thickening of the interlobular septa, of the peribronchial and periarterial connective tissue and of the subpleural layers and from a collapse induration of the alveoli. The small groups of alveoli which remain in the sclerosed areas are often filled with alveolar phagocytes stuffed with coal pigment. Organization of alveolar exudate may add to the obliteration of the lung parenchyma. The walls of the bronchi are thickened and the mucosa is infiltrated by round cells and plasma cells. The medium sized and larger arteries and veins show varying degrees of intimal proliferation.

In the tracheobronchial lymph nodes nodules similar to those in the lung are formed. They are particularly rich in coal pigment and are first seen in the cortex, in the region of the secondary follicles. Later, they extend into the medulla (Giese). In cases of slight silicosis there may be only a diffuse sclerosis of the reticulum without nodules.

In asbestosis the characteristic nodules are less well defined than in silicosis. In some cases they may be entirely absent (McDonald). An extreme, universal fibrosis dominates the microscopic picture (Lynch and Smith). There is a diffuse interstitial pneumonia or complete obliteration of the alveoli. The golden brown asbestosis bodies are present in great number and can be found in the lumen of the alveoli, bronchioli and bronchi and in the areas of less advanced interstitial fibrosis. In the lumen of the alveoli many of the bodies are surrounded by foreign body giant cells. Lynch and Smith described a golden brown, granular pigment suggesting disintegrated asbestosis bodies.

THE RELATION BETWEEN SILICOSIS AND TUBERCULOSIS

The silicotic fibrosis makes the lungs very prone to infections and it is especially the tuberculosis that follows the silicosis like a shadow. Gardner says that more than 75 per cent. of the people who contract silicosis die of tuberculosis which may manifest itself many years after the exposure to the dust has ceased. Simon, Strachon and Irvin, who studied a severe form of silicosis common among South African gold miners, distinguish between simple silicosis and infective silicosis of which tuberculo-silicosis is the most important form. Under the influence of the tuberculous infection the silicotic lesions change their appearance. The tuberculo-silicotic nodules are larger than the simple silicotic nodules (Mavrogordato). They fuse together and in the center there are evidences of necrosis which may be very slight (Mavrogordato, Simon and Strachon). Besides the mixed tuberculo-silicotic nodules simple silicotic nodules are often present and about these nodules tuberculous granulation tissue may develop.

The most striking effect of the tuberculosis upon the silicosis is the formation of large areas of dense sclerosis in which the nodules become obscured. Thus, the picture approaches that which has been described in the preceding chapters as advanced, nodose form of silicosis. Many investigators, therefore, maintain that this advanced, nodose form is always tuberculo-silicotic in nature (Watkins-Pitchford, Mavrogordato, Siegmund, Gross, Ickert, Huebschmann, Husten, Policard, Scheinin, Gardner, et al.). Admitting the importance of infection in determining the nodose character of advanced silicosis it remains to be proved that this infection is always tuberculosis. I saw several cases of advanced nodose silicosis which had died from

decompensation of the hypertrophic right heart and which on careful microscopic examination failed to show any evidences of tuberculosis (see also Giese, Bergstrand, Berg, and others). The positive biological test for tubercle bacilli is of doubtful value since tubercle bacilli have been demonstrated in normal lungs and lymph nodes of hospital patients. According to Gardner granite dust with a free silicon content of about 35 per cent. produces nodules only in the presence of a tuberculous infection.

Since the relations between tuberculosis and silicosis are so intimate that a type of lesion results which does not occur in the uncomplicated form of the two diseases tuberculo-silicosis has often been considered a disease sui generis. Anatomically the differences between tuberculo-silicosis and other forms of benign, sclerosing pulmonary tuberculosis are rather quantitative than qualitative and in a doubtful case the examination of incinerated sections under the polariscope will have to decide whether this case is silicotic or simple fibrotic.

The stimulating influence of the silicosis upon the tuberculous infection is well illustrated by animal experiments of which those of Gardner are of particular interest. Gardner used a strain of tubercle bacilli which was so little virulent that it produced in guinea pigs a regressive, nonfatal disease. If this strain was injected into guinea pigs that had previously been exposed to prolonged dusting an extensive pulmonary tuberculosis with involvement of the abdominal viscera developed which caused the death of the animals. By reinoculation of the strain into normal animals it could be shown that the strain had not increased in virulence. Kettle found that in animals injected with colloidal silicon the tubercle bacilli proliferated much more luxuriously than in normal controls. I do not think that it is necessary to assume a direct influence of the silicon upon the tubercle bacilli or immunological phenomena (e. g. absorption of complement) in order to account for the effect of the silicosis upon the tuberculosis. The local tissue damage caused by the silicon crystals is apparently sufficient to favor the spreading of the infection.

The silicosis may affect a lung which has been the site of a mild tuberculosis stimulating it to more active progression. In the majority of the cases the tuberculosis is added to the silicosis (addition tuberculosis of Redeker). This addition tuberculosis may result from the endogenous exacerbation of a latent focus, especially in a lymph-node, with hematogenous dissemination to the lungs, or one may be dealing with a new, exogenous superinfection. According to my opinion the later mechanism is of greater significance. This exogenous superinfection accounts for the differences in the frequency of tuberculo-silcosis in different localities. If a few cases of open tuberculosis are scattered among people exposed to silicosis the infection is apt to spread rapidly, in particular under poor hygienic conditions.

The classical form of tuberculo-silicosis is a relatively benign, slowly progressing, isolated pulmonary tuberculosis with much scarring. The tuberculosis intensifies the sclerosing effect of the silicosis while the silicosis favors the fibrotic involution of the tuberculous lesions. In the terminal stages exudative-caseous and ulcerative lesions may develop (Eickenbuch and others). In acute silicosis, Gardner found a predominance of exudative caseous processes.

In asbestosis the predisposition to tuberculosis seems to be less marked than in silicosis. Wood and Gloyne found in a series of 57 cases of asbestosis 10 complicated with tuberculosis. Of these ten cases two succumbed to the tuberculosis. The tuberculous lesions were caseous in character with little connective tissue formation and giant cells were scanty. Asbestosis bodies could be found in the caseous areas side by side with clumps of tubercle bacilli. Gardner and Cummings found destruction of the asbestosis bodies in the caseous areas.

THE RELATION BETWEEN SILICOSIS AND CARCINOMA OF THE LUNG:

In recent years the apparent increase in frequency of pulmonary carcinoma has caused much speculation and reference has repeatedly been made to dust as a possible pathogenetic agent. There are, however, no indications that silicosis predisposes to carcinoma of the lung (Hardfield and Garrot, Strachan, Saupe, Ickert, Schulte, di Biasi and others). In 90 cases of primary carcinoma of the lung which I have examined at autopsy during the last few years there was not a single case with anatomical evidences of silicosis. Silicosis does not cause excessive epithelial proliferation or epithelial metaplasia which so often precede the development of a carcinoma.

Schmorl referred to scars from perforations of indurated lymph nodes into bronchi as source for carcinoma. As pointed out in a preceding chapter these perforations are due to tuberculosis and not to silicosis.

The great frequency of malignant pulmonary neoplasms among the miners in the cobalt mines of Schneeberg and the radium mines of Joachimstal cannot be related to silicon (Saupe). After excluding cobalt and arsenic Saupe comes to the conclusion that the high radium emanation in these mines is of greatest importance. In the course of 15 years the intake of radium chloride is estimated at about 55 mg. The carcinogenic effect of radium also is shown in industries using radioactive substances.

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RADIOGRAPHIC VISUALIZATION OF FIBROSIS PRODUCED BY DUST INHALATION

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In the studies of disease entities we have almost always started our investigations with the terminal stages and worked backward to their etiological origins and early pathological processes. Consequently the widest dissemination of facts and most voluminous writings have concerned the advanced or moderately advanced stages of most diseases. This is true because the clinician is the first to meet and attack a disease as a clinical entity and his description of its course, symptoms and physical findings is the foundation upon which theories and facts must stand. He needs little or no help to diagnose advanced or moderately advanced stages of disease but incipiency or very early pathology is usually most difficult or impossible to recognize without aid or confirmation of laboratory procedures.

Pneumonoconiosis is no exception.

It is my belief that any dust of such fineness as to reach the alveoli, and breathed in sufficient quantities for a long enough period of time, will produce some visible degree of lung fibrosis on the radiograph. Silica dust produces the most dense fibrosis. The pathologist has traced the progress of the dust particles from the alveoli, by phagocytosis into and through the pulmonary lymphatics, and described their deposition in

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lymph spaces and nodes, where by irritation they produce either the linear or nodular fibrosis seen on the film.

As pneumonoconiosis reaches a rather advanced stage before it presents clinical symptoms and physical findings sufficiently prominent for diagnosis, radiographic visualization must be relied upon for the early pathology. This is particularly true if any attempt is to be made to prevent or arrest the progress of the disease.

In all occupations where dust inhalation may be a hazard, it is advisable to radiograph the chest before employment and to determine the extent of lung fibrosis. Individuals with extensive fibrosis or other pulmonary lesions should not be employed in dusty occupations. All persons working in dust producing operations should be radiographed every six months to discover early lung changes and to serve as a check on preventive measures.

Serial radiography is as necessary for the prevention and control of pneumonoconiosis as it is for tuberculosis.

Pneumonoconiosis, according to the progress of the disease, is usually described in three stages, namely, first, second and third. The clinical, pathologic, and radiographic findings coincide to a remarkable degree. The increased radiographic densities are due to increased fibrosis and not to shadows cast by dust deposits.

In the first stage the radiograph shows an increase of the lung root shadows beyond normal limits due to enlargement of the lymph nodes and thickening of the larger bronchi and vessels. The branchings of the bronchial and vascular trees appear more numerous and are wider and denser than normal. As the pleura is approached the size and density of the shadows tend to decrease. The spaces between the more prominent markings, which in the normal lung are usually clear, show many fine linear shadows. The markings of this stage are all linear in character; no mottling is seen and adhesions are rare. The condition is bilateral, with a fairly even distribution in all lobes; however, the right lung or the lower lobes may show the greattr advancement, due to the greater amount of air going to these locations. Passive congestion and chronic infection may produce somewhat similar changes, but the markings are not usually so clear-cut nor so definitely symmetrical as are those of dust pathology.

In the second stage, the hilum shadows and linear markings of the broncheovascular trees usually show further increase in size and density, but, in addition, there are seen many small, round, discrete densities which produce the effect of generalized mottling on the film. The mottled appearance is due to fibrosis of the many lymphoid tissue deposits of the lymphatic system. The discrete shadows vary in size from about one millimeter to ten millimeters in diameter, the larger being partly due to coalescence of smaller nodules. The nodules show a fairly even distribution throughout both right and left lobes but are usually more numerous in the right. The pleura is thickened in some areas, and the diaphragm, particularly the inner portions, may be irregular and roughened from adhesions.

In the third stage the extensive generalized interstitial fibrosis displaces the more or less discrete and sharply defined markings of the first and second stages. The small discrete nodules of the second stage coalesce to form larger irregular masses which, on the radiograph, produce the "snow-storm effect," characteristic of this stage. If the fibrosis continues, with further confluence of the larger densities, massive consolidation is produced. The consolidations are usually bilateral, and in extent may involve any part of a lobe or an entire lobe, most often the upper. This stage shows more extensive pleural thickening and adhesions, particularly to the diaphragm, often causing marked elevation and irregularity. The heart and large vessels are pulled toward the right side and often occupy a vertical position in the thorax.

The existence of pneumonoconiosis establishes a predisposition to intercurrent respiratory infections, and tuberculosis is often a complicating factor. Tuberculous infection may become engrafted on dust pathology during any stage, but most usually in the second or third stages.

As its presence is frequently a difficult matter to determine from the radiographic film alone, a few differential points will be noted here. Pneumonoconiosis is bilateral, symmetrical, affects all lobes, the shadows are clear-cut, well defined and hard in appearance, the apices are usually clear until far advanced, and no cavitation occurs. Tuberculosis is ordinarily unilateral, not symmetrical; the apices become involved early; it is generally confined to the upper lobes; the shadows are not so clear-cut or well defined and

are softer in appearance, and cavitation occurs with advancement.

CONCLUSIONS

In conclusion, I wish to emphasize the following facts:

1. Inhalation of dusts is essentially an industrial hazard, many and varied occupations presenting it.

2. So far, silica dust has been found to be most harmful, but other dusts produce lung changes to a lesser extent if conditions are suitable.

3. Only very fine dust particles, less than 10 microns in diameter, reach the alveoli and cause pathology.

4. The changes produced are fibrosis of the lymphoid deposits and lymphatic drainage system.

5. The radiograph presents a definite picture of the progress of the disease. It is the easiest, surest, and often the only method of diagnosis, and we should, more generally, interest ourselves in further study of this condition.

DISCUSSION ON SYMPOSIUM ON PNEUMONOCONIOSIS

Dr. Don C. Sutton, Chicago: When it was first decided we should have a symposium on pneumonoconiosis, apparently the reason back of it was the legal difficulties that have developed during the last three or four years.

It was intended that there should be a lawyer on this symposium. In looking over the field of men who were qualified to discuss the subject, we were unable to find anyone who really knew something about silicosis, except men who were employed by the larger industrial concerns, and it seemed to be impossible to get a completely unbiased legal opinion.

It was rather interesting in talking to some of these lawyers to learn that after all they look at it with a sense of fairness, that they feel the industry has a certain liability so far as the laborer is concerned, but that their greatest difficulty is the fact that no two doctors agree on the findings in a particular patient or on the diagnosis. So they are always able to get expert testimony on both sides of the question.

We have heard very well discussed this afternoon, first by Dr. Sappington, the study of the hazards in various types or trades so that apparently this is the point which both industry and we as doctors are interested in—that of prevention. And certainly industry is just as much interested financially in preventing silicosis as we are purely physically.

Dr. Head and Dr. Flinn have covered fully the field of diagnosis, with Dr. Jaffé, of course, completing the pathological picture.

We still are left without knowing how to judge the patient that we see, and I think most of you are in the some position I am in practicing medicine. We want to know how sick our patient is, how great his disability is, and possibly his future.

I think I would find it very hard to get on the stand and testify fairly to either side with my present knowledge of pneumonoconiosis. There are certain factors though, I think, that offer a chance of evaluating the amount of disability.

As Dr. Jaffé has said the circulation is seriously impaired. As a result of this destruction of circulation there is an increased load on the right side of the heart. Another factor is the incomplete aeration of the blood. The fibrosis of the lung itself reduces greatly the normal elasticity of the lung so that we find these patients have a greatly reduced vital capacity.

There are certain studies that point towards the fact that when the tidal air is increased up to forty per cent. above normal, there is a definite decrease in the amount of oxygen in the blood. It is also quite definitely known that a decrease of the oxygen in the blood to eighty-five per cent. or ninety per cent. of normal causes some cyanosis and definite disability of the patient. So that we do have some tests that I think could be used to evaluate somewhat the amount of dysfunction that the particular patient has.

I am talking, of course, entirely of uncomplicated cases as Dr. Head and Dr. Jaffé both called attention to the fact that complications do tend to occur—bronchitis, secondary infections, and tuberculosis. But for the uncomplicated case, the physical examination and the routine x-ray does not give us an adequate picture of measuse of the patient's disability.

It seems to me the study of vital capacity, tidal air and of oxygen consumption does give something to go on.

Wm. D. McNally, Chicago: The theory of silicosis being caused by colloidal silicate has probably been now exploded. At the time Guy and Kettle gave that theory to the people they were not acquainted with the French literature regarding the presence of fluorides in the blood. Sodium fluoride has great affinity for the silicon dioxide molecule, and in the extremely small size that the silicon is present in the lungs, this sodium fluoride tries to establish an equilibrium just the same as sodium bicarbonate does in our blood stream.

The silicon dioxide is acted upon chemically by the fluoride and forms a silicon tetrafluoride. This silicon tetrafluoride by its toxic action causes the fibrosis that you have seen.

In another month or so we will have ample proof of that for you as we are working upon that phase of silicosis.

Dr. Jaffé does not think much of the chemical theory of silicosis. I disagree with him. Of course I have worked more on the chemical side than Dr. Jaffé has.

Silicon dioxide is a normal constituent of every tissue in the human body. We find it in the fetal lung from six months up. It is found in babies' hair, it is found in the nails of the adult, it is found every place in the human body.

We have run several hundred lungs and I have

searched the literature quite thoroughly—German, French and English—and all investigators agree that there is a normal silicon dioxide content in the human lung. That is close to 1.5 mg. per gram of dried tissue. Occasionally we have found up to 1.8 and 1.9. Therefore, anything that is over two milligrams per gram of dry tissue, shows an exposure of dust, and a pathologist cannot prove that. It takes a chemist to prove it.

I want you to understand that. There is no way that I can demonstrate by sections, Dr. Jaffé, or anybody else, that a lung has more than one and a half milligrams of silica per gram of dried lung. When it reaches three and nine-tenths milligrams per gram of dry tissue, and that is the smallest amount where I have found a beginning fibrosis, it can be noticed microscopically. It is nearly always over five before this can be demonstrated. The German authorities say seven, but we have found it several times when the silica was a little over five milligrams. So we can determine definitely and quantitatively, what you cannot do in pathology, the amount of silicon dioxide in that lung, and the damage. We know when there is 5.2 milligrams per gram of dry tissue there is damage to that lung. When it gets up to eight, there is a lot of damage. When it gets up to twenty-nine, we still know more about it.

Take these granite workers in Vermont. The lungs I have examined ran up to 29. We know that lung is all shot to pieces. There is no lung there hardly at all, so I want to take exception that the chemical phase of silicon dioxide should not be ignored.

One of the speakers said that washing powder was silicon dioxide, or contained large amounts. Technically that is wrong. Dr. Head, silicon dioxide is not in washing powders. It is in scouring powders and scouring bricks, so just change that wording in there, because maybe the ladies will not use washing powder.

When Dr. Sappington correlates all of his data he should correlate the data of the pathologist along with the chemist and then you will have a clear picture of what silicosis is.

EASIER TRANSFUSIONS FOR THE GENERAL PRACTITIONER

F. J. Otis, M. D., F. A. C. S.

MOLINE, ILL.

When a general practitioner recommends a transfusion and it becomes his duty to perform the operation, it is a very important matter. He must deliver one hundred per cent. He must not fail. Otherwise the life of the patient is on his shoulders. Once undertaken he cannot sidestep nor postpone the responsibility. The ignominy of a failure is all his.

Therefore, out of the wealth of medical history I wish to call your attention to those points of medical art which have given us these ten

PROCEDURE IDEALS

1. Needle in the vein without operating.
2. Closed aseptic method.
3. Undiluted blood gives fewer reactions.
4. Small container so blood passes over quickly.
5. No loss of blood.
6. No back flow to the donor.
7. Saline or Ringer's solution ready for patient's or donor's need.
8. Time even when a slow transfusion is necessary.
9. Time to change donors if desired.
10. Time to reset needle when necessary.

The nearly three hundred years from transfusion records of old Pepys' diary past the impellers, cannulas, and quills to Crile's intima to intima cannula of 1898, gave us little of value because we had not yet come to the great discoveries which were necessary to make blood transfusion practical and possible. It did tell us one thing we have largely forgotten about embolism from injected air. That is that an amount below twenty cubic centimeters might not be harmful.

THE GREAT DISCOVERIES[1]

1. 1890: Slow clotting of blood in a vein or vessel. (Transfusion by suture).
2. 1899-1911: Agglutination leads to typing. (Transfusion becomes safe).
3. 1895-1911: Oil and paraffin coatings slow clotting. (Kimpton-Brown, Percy, Vincent tubes).
4. 1915: Citrated blood transfusion by Lewisohn[2] and Weil. (Standard method of the Great War).
5. 1914-1924: Intravenous drip popularized by Mates[3] keeps needles open for days.

Inability to measure the blood and the necessity of an operation has lessened the value of the first great discovery.

Typing was the second and key discovery for transfusion success. Incidentally, it tells why the homeopathic syringe dose of the nineties oft repeated was more successful than the calamatous allopathic doses of blood. Today, in the presence of some of the subagglutinins, it is far safer

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to give a repeated small dose than the single large dose now so easily accomplished.

According to Mason[4] seventy-four per cent. of our hospitals are using the Moss classification. As an international classification is coming forward, recommended by Landsteiner, the discoverer of agglutination, we should keep before us their relationships.

1927 International	O	A	B	AB
1907 Jansky	I	II	III	IV
1910 Moss	IV	II	III	I
Percentage of occurrence	*45*	*42*	*10*	*3*

The practical use of Moss' typing was scarcely under way, and the Crile and Carrel successful transfusions were but a few years past when Curtis and David[5] brought forward the third great discovery, the use of paraffin coating to delay clotting. Oil had been used in needles and cannulas since 1895, but paraffin was more suitable for tubes. In 1913 Kimpton-Brown[6] popularized the method, assisted in the same year by Percy[7], and later Vincent.

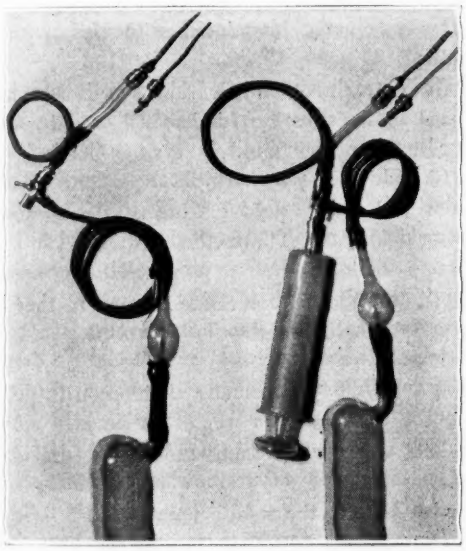


Fig. 1. A simple direct method.

Hardly had these methods gotten under way when the fourth great discovery came forward in 1915 with its simplicity. It required no operation to get the donor's blood. Citrated blood kept for hours, and even days. The Great War demanded a simple method, so under the advice of Bernheim the Lewisohn-Weil method of citrating the blood with first 0.2 per cent. and later 0.25 per cent. was made the standard. The open method of collecting the blood was unpleasant to the artistic surgical technician, so a closed method was included in the War standard.

Thus modified, this method had all the procedure ideals except elasticity of drawing. Often one's needle clotted with only a portion of the desired dose drawn. Apparently this need was early anticipated by Hartman[8], a young army officer who devised a method and later made a special needle to admit citrate to the blood as it flowed out the needle into the tubes. The collecting tubes were thus kept clear of coagulation. The momentary pinching of the collecting tube would flush the citrating solution into the needle. This procedure made the citrate method meet all our ideals. Later Doctor Pond[9] rediscovered and reemphasized the importance of needle citration. Doctors Cowles and Antz reported a device to accomplish the same purpose in a more complicated manner, but possessing the far more ideal gravity principle.

Having overlooked these reports, I exhibited here a year ago a B-D Y observation tube to admit the citrate after leaving the needle.

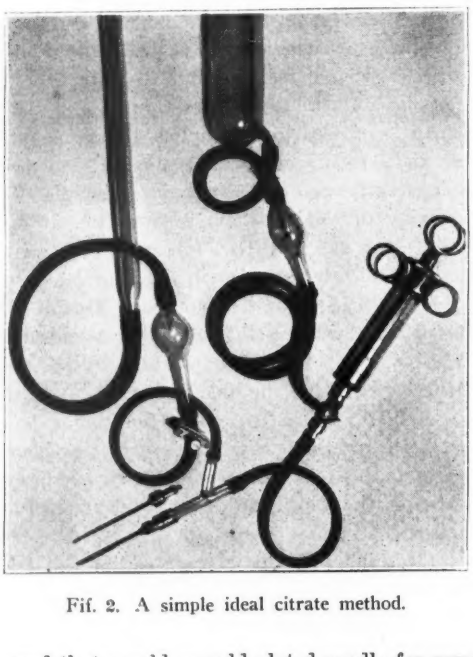
A Simple Ideal Citrate Method. Permit the 2.5 per cent. citrate solution to pass a bulb drip indicator and Hoffman clamp into the Y observation tube, citrating the blood just after it leaves the needle. Use a ten cubic centimeter local anesthetic syringe to aspirate the citrating blood, and by means of a salvarsan three way stopcock discharge the blood up into a salvarsan tube from which it may pass to the recipient by gravity. It might even be transfused immediately. The needle is automatically flushed with citrate while discharging the blood from the syringe. A recent improvement is the slowing of the flow to the patient, using a closed drip bulb to observe the rate of flow.

A five hundred cubic centimeter dose of blood would require 1.25 grams of sodium citrate. The body takes 5 grams without harm.[11]

Because of citrate reactions it is well to note some experimental work on citrate toxicity being done at Vanderbilt University.[12] It suggests when the calcium content of the blood has been depleted, as it often is in patients requiring transfusion, a smaller dose than 5 grams may be toxic. Possibly there may lie the reason why the citrate method gives double the reactions noted with the whole blood methods.

While I regard the intravenous drip, popularized by Matas, as the fifth great discovery, flushing really was introduced to transfusion, for maintaining the cannulas open, by Satterlee and Hooker in 1914.

The value of their suggestion was lost in the hurry of the Great War. They applied it to the most needy method, the paraffin tube method. They used a very small paraffined container, and frequently passed from donor to recipient, fulfilling the ideal which demands but a small amount of blood out at a time. Unfortunately they used special gold cannulas, which I suspect prevented general use, for I have observed that a gold or gold-plated needle for some reason has about five times the drag of a poorly polished steel needle passing through the tissue.



Fif. 2. A simple ideal citrate method.

Even today the paraffined tube method needs a rediscovering of their methods to add elasticity in securing the blood.

When a surgeon operates on a desperate case, it is well to be also ready to transfuse blood. Doctor Collins of Peoria says: "If you anticipate the need of fluids after an operation, it is well to start the hypodermoclysis before the operation." Too frequently when you need transfusion, most of it cannot be accomplished in time.

The Matas drip discovery, familiar to all surgeons, makes possible

A Simple Transfusion Method. First put a three way stop-cock in each of two Matas intravenous drip outfits to control the drip. Connect one to the patient to be operated on, and the other to a suitable donor. Then at any time during the operation an assistant can draw any desired amount of blood from the donor and inject it into the patient through the three way stop-cocks. He can repeat when necessary. It makes a very simple whole blood transfusion, always under control; and due to the drip a far more elastic method in reference to time than the non-drip syringe methods.

Of the two methods to keep the donor's needle active, the flushing and the oiled obturator, the latter was early introduced with the syringe method.

While small amounts of blood had been injected in the nineties, Lindeman[13] brought forward an efficient multiple syringe method in 1913. He used the sharp obturator in his cannulas and a thorough flushing of the syringes between fillings. The method required two operators, besides a nurse to wash the syringes, but it was the most effective method in that it gave eventually the smallest percentage of reactions.

If you follow Lindeman's instruction in placing the cannulas, you would not have so much trouble today while using the needle, provided it contained an obturator that could be extended beyond the needle point.

To keep the needle in place some have fastened it to the skin with a cambric needle or adhesive plaster. Others have placed a rubber tube between the needle and point of attachment to lessen the consequences of a false motion while attaching.

The syringe type of transfusions have followed two lines of development to meet our ideals. They are the direct syringe and the syringe and stop-cock combination.

Doctor Strauss[14] followed the Lindeman multiple syringe method. He used an obturator to keep the needle open, the Watson[15] cambric needle, popularized by Pemberton[16], to secure the vein, and another cambric needle to fasten the transfusion needle securely to the skin. He used three one hundred cubic centimeter syringes.

The method meets most of our needs and most of our ideals.

However, the stop-cock controlled syringes, started by Unger,[17] have met our ideals most perfectly. While Brines[18] modified it to a straight non-flushing two syringe apparatus, he exchanged its elasticity for speed. To keep this improved speed, Martin[19] had to put in an extra three way stop-cock to admit flushing solutions, and thus regain Unger's former elastic qualities.

Where the blood syringe is depended upon also to flush the system, one is lost when its blood film coagulates and stops it. It is then that the value of the gravity drip, to keep the needles of the patient and donor open, becomes apparent.

Some of the excellent modern instruments turn a valve or rotate a plunger to fill and empty. They also have a mid position for flushing. There are others possessing very simple action. They have just a "push and pull," or "turn a crank" that operates them so very automatically. To do so, however, most of them have sacrificed the possibility of flushing.

While Martin made a two man double syringe out of Unger's, James made a one man non-reversible, continuous flow, double syringe apparatus that should have had more users. I suspect its lack of popularity among the general practitioners was due to the need of the flushing principle. Morse introduced a stop-cock near each needle to pump saline into the tubes. But the syringes when congealed could not flush the needles, and it was also necessary to make the device temporarily reversible to flush to the donor.

After trying a non-flushing, continuous flow, double syringe I undertook its improvement by following the Martin and Unger modification, and introduced two stop-cocks instead of their one. This provided for drips to each needle.

With that instrument I could transfuse a small amount every ten minutes for an hour or two if I desired. It gave me the full extreme of elasticity.

When one observes the placing of the needles and the use of the thumb to prevent loss of blood, and sometimes notices the contamination of the needle, one wishes there were cover lids or caps on the obturators to protect the end of the needle.

I have used a Luer-Lok rubber tube adapter (B-D H/468L) as a cap to protect the needle and serve also as a handle while inserting the needles. It would also hold a wire obturator back while inserting until time to advance it beyond the point in the vein.

Allow me also to call attention to Brooks' art in using the needle for the Kimpton-Brown method. He says after the needle is in the vein compress the vein over the needle point with the ring finger. Then withdraw the obturator and there will be no loss of blood while you connect up. Do the same in disconnecting.

The Ideal Instrument

1. Continuous flow saves time.
2. Standard replaceable syringes.
3. Operates by a simple to and fro motion.
4. One way flow not reversible.
5. Locked against back flow.
6. Easy finger assembly.
7. No loss of essential small parts.
8. Permits saline drip to each needle if desired.

If you have an instrument that meets these requirements, then blood transfusion will be so easy it becomes dangerous without noting the following:

Safety Procedures

1. Typing checked by cross matching the bloods.
2. In case of reaction or chills, stop!
3. Transfuse only conscious patients to avoid chill.
4. Dose under 350 cc. caused no type reaction deaths.[20]
5. Donors on four hour fasts to avoid allergy.
6. Keep ready epinephrin hypodermic to treat reactions.[21]
7. Keep hypodermic of calcium gluconate ready for depleted calcium patients.
8. Intravenous salt or Ringer's solution set up for shock.
9. Immerse new rubber tubing thirty minutes in normal Na OH solution before washing.[22]

DISCUSSION

Dr. John J. Pflock, Chicago: Dr. Otis has covered the instrumentation and methods very completely, so there is hardly anything to say about the instruments to be used. I think the simpler the instrument we use the better it is. It requires a skilled hand to do a blood transfusion and oftentimes we delegate this operation to inexperienced hands. It is essential that the one doing

a blood transfusion should be skilled and carefully trained. Very often internes and even technicians, possessing neither skill nor experience, are allowed to do blood transfusions.

The Scannel set with Unger type needles has given me the best service, not expensive, almost foolproof, and simple to operate. I find the reaction following transfusion with unmodified whole blood much less than that of the citrated blood. The use of whole blood is most satisfactory because none of the blood is destroyed by citration. Checking up on the typing is very important and should not be done hours or days before the transfusion, but almost immediately before transfusion is contemplated. The donors also should be carefully checked up to see that we do not get diseased blood. We prefer the middle aged, healthy, Wassermann negative male as a donor.

The advantages of using whole blood are that first of all we get the blood as is, blood without any foreign bodies in it. The operation is done quickly and very safely. There is no contamination of the blood in this method and fewer reactions result from whole blood than from citrated blood. We get less chilling in whole blood than in citrated blood, and the blood platelets are not disturbed or destroyed.

The indications for blood transfusions were discussed. I believe any patient who has a blood count less than three million and a hemoglobin less than 45 is entitled to a blood transfusion, no matter what the other indications are. One often hesitates whether or not a patient should have a blood transfusion. My rule is to do as with a sore throat that suggests diphtheria. If I am in doubt whether or not to give the patient antitoxin, I give it. The same with a blood transfusion, when in doubt, transfuse.

Dr. Frank J. Otis, Moline (closing): I agree with Dr. Pflock entirely. It occurred to me that simplicity of action might require one or two more things. As I tell my nurses, be ready so as to move right off on anything that is necessary. By pressing on a valve, as you push the lever to fill the syringe, you admit the citrate to the syringe; continue moving the lever the blood starts to the donor, and by stopping the lever in a middle position it establishes a drip to each one. We do not have as many assistants and internes, in our work in the country, and if I am working with this apparatus and there comes an emergency in the delivery room, I can stop the syringe, go down to the delivery room, return in five to ten minutes and proceed with the transfusion as though nothing had happened.

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SURGICAL RELIEF OF PAINFUL DEGLUTITION IN LARYNGEAL TUBERCULOSIS

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The early diagnosis and treatment of tuberculosis of the larynx must assume a prominent role in any anti-tuberculosis program. The delay in early diagnosis results in the loss of much valuable time before the proper treatment can be instituted, thus lowering the patient's much needed strength, increases the dangers of complications, offers a poor general prognosis, and lessens the chances for the patient's final recovery.

Considerable data have been presented on the various measures for the prevention of laryngeal complications, and there have been many contributions in the literature on prophylaxis in the early management of laryngeal tuberculosis, but comparatively few observers have made mention of surgical treatment of the sensory nerve of the

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larynx to afford early relief from pain on swallowing in any stage of this much discussed and dreaded complication.

Only since Hoffman[1] introduced the blocking of the superior laryngeal nerve by the injection of a strong alcohol solution, has it become possible to relieve the pain in tuberculosis of the larynx for any appreciable length of time. Unfortunately the success of the injection varies, as the duration of the anesthesia and the relief of pain fluctuates from a few hours to months or more. A further disadvantage of this method is the fact that after the first injection all subsequent injections are less likely to be successful, because of the formation of connective tissue. As recorded in three of our cases and again called attention to by Nordland[2] there is the possibility of paralysis of the tongue, due to the effect of the alcohol extending into the hypoglossal nerve so closely related. Shukoff[3] states that this anatomic condition is found particularly in short necked individuals. Shugt[4] records a number of cases treated for dysphagia by the injection of alcohol in which a mild edema on the diseased side following the injection was recorded. Boncour[5] is the only observer who records a recession of the inflammatory condition after the blocking of the nerve by the injection of alcohol.

It may be well at this point to describe briefly the technique of the foregoing procedure. When the superior laryngeal nerve is injected, the usual thorough asepsis should be followed. The fluid for injection is 2 drachms of essentially an 85 to 90 per cent. alcohol to which 1-3 drops of pure phenol has been added. A glass hypodermic syringe with a 1½-inch twenty-four gauge needle is used. The patient may lie on his or her back, or may sit upright. The superior cornu of the thyroid cartilage and the greater cornu of the hyoid bone are located by palpation with the index finger and thumb. The needle point is plunged through the skin at an angle of 45 degrees into the thyrohyoid space for a distance of about 1 cm. The point of the needle is rotated until the patient complains of a sharp pain in the ear on the same side. This indicates that the internal branch of the superior laryngeal nerve has been touched. About one or two c.c. in quantity is injected. If the patient coughs, it indicates that the needle point has penetrated into the larynx. This causes no harm, but the needle point should be withdrawn a very short distance and the injection continued.

Having presented the technique and the disadvantages of the nerve injections, we are pleased to call attention to a safe surgical method of relieving the patient quite early and permanently from pain on deglutition, by resecting the sensory nerve to the larynx, the superior laryngeal; this makes the patient's waking and resting hours bearable, prevents interference with the patient's nutrition, comfort and rest, that are so necessary toward maintaining vital forces of the body.

It should be clearly understood that surgical procedures are not intended to replace non-surgical methods entirely, but are to be considered as adjuncts to those methods. The failure heretofore to make full use of all available therapeutic measures has frequently been due to the prevailing indifference to surgical measures, to too technical discussions, or to the lack of general information as to the ever increasing indications for surgical intervention in the management of laryngeal tuberculosis.

Here we wish to record our experience and to call attention to the value of sectioning the superior laryngeal nerve in cases of tuberculosis of the larynx. This method was first resorted to by Avellis in 1909 for relief of dysphagia in laryngeal tuberculosis. This operation has no contraindications, is exceedingly simple to perform, causes little or no shock, and is absolutely safe.

Before the operative technique is discussed, it is well worth remembering that the superior laryngeal nerve arises from the middle of the ganglion of the trunk of the vagus and in its course receives a branch from the superior cervical ganglion of the sympathetic. It descends laterally to the pharynx behind the internal carotid artery where it divides into the two branches, the external and internal laryngeal nerves. The external laryngeal branch runs beneath the sternothyroid muscle to supply the cricothyroid muscle; the internal branch passes down to the opening in the thyrohyoid membrane through which it passes with the superior laryngeal artery and is distributed to the mucous membrane of the larynx. The branches to the mucous membrane are distributed to the anterior portion of the epiglottis, the base of the tongue, and the epiglottidean folds; while others pass backward and supply the membrane which

lines the cavity of the larynx as low down as the vocal cords.

The operative technique for the section of the superior laryngeal nerve is as follows: the skin is prepared in the usual manner and infiltrated with several c.c. of a one per cent. novocain solution at the site of the intended incision. An incision, two or three inches long, is made through the skin and cervical fascia, extending from the middle of the thyroid cartilage, running parallel to the greater cornu of the hyoid bone to inner border of the sternocleidomastoid muscle. The omohyoid muscle is pulled forward toward the mid-line while the sternocleidomastoid muscle is retracted backward where the large vessels of the neck are carefully avoided. The lateral end of the hyoid cornu is now palpated and by blunt dissection the superior thyroid artery is traced. Superior and anterior to the loop of the superior thyroid artery the superior laryngeal nerve can be seen lying in the thyrohyoid membrane where the internal branch is picked up and sectioned between two forceps.

The cases operated upon in our series were all far advanced cases of pulmonary tuberculosis complicated by tuberculous laryngitis.

In all, seven cases were operated upon and nine sections performed, as in two of our cases bilateral sections were made as recommended by Wachman.[6] Every case responded immediately with relief from pain on swallowing; in a short time there was a decided general improvement with increased strength and resistance brought about by the increased rest and the ability to enjoy more nourishment. Following the operation mild motor disturbances were complained of for several days in two cases and then promptly subsided. In those in our series in which we had to contend with extensive ulceration of the larynx we did not notice any changes in the larynx following resection of the superior laryngeal nerve, but in four non-ulcerative cases of our series we did note a lessening of the infiltration. In two of our cases there was a persisting complaint of pain on swallowing in spite of a successful severance of the nerve, due in our opinion, to the extension of ulceration, perichondritis and chondritis, and not related to the innervation of the superior laryngeal nerve.

Numerous authors assert that resection of the superior laryngeal nerve has a healing influence on the tuberculous process of the larynx. Mayer[7] observed the disappearance of the acute inflammatory symptoms which were complicating the tuberculosis of the larynx. He states that the inflammatory conditions are produced by the accumulation and stagnation of mucus and pus over the larynx and the pyriform sinuses. This material is either coughed up or swollowed because of the pain on swallowing and irritation and inflammation ensue. Teller[8] records a definite recession of the infiltration in a series following sectioning of the superior laryngeal nerve. Muskat[9] in his series of cases observed a velvety, dusky red hyperemia following resection with subsidence of the infective process. Jackson and Coates[10] state that a transverse section of this nerve is followed by a loss of sensitivity in the laryngeal mucous membrane, a relaxation of the thyrocricoid muscle with a consequent lowering of the pitch and a diminution in the clearness of the voice. Kolina[11] states that the resection of the superior laryngeal nerve has a direct healing influence on the inflammatory condition and consequently on the tuberculous process of the larynx. As a result of our own observations, as well as those reported by other workers, we are firmly convinced that resection of the superior laryngeal nerve for dysphagia in tuberculosis of the larynx is our choice over any of the other recorded available methods more commonly used today, because of the simplicity of the operation and the certainty of permanent relief that it affords.

CONCLUSIONS

1. There are no contraindications to sectioning the superior laryngeal nerve on one or both sides in tuberculosis of the larynx where there is pain in swallowing and not relieved by simple conservative measures.

2. In our series we did not observe any unusual or lasting complications.

3. The operative technique is simple, rapid and is absolutely safe and we plead that this operation be performed early so that the patient can be kept from exhaustion because of pain that interferes with proper nutrition.

4. It definitely affords the patient permanent relief, helping to restore his resistance and much needed strength. We firmly believe that resection of the superior laryngeal nerve in cases of laryngeal tuberculosis deserves a place in our surgical armamentarium because of its wide-

spread applicability to warrant general consideration and sympathetic use.

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DISCUSSION

Dr. Louis Savitt, Chicago: It is very gratifying to hear Dr. Fishman talk of the work performed by himself and Dr. Lederer. I have had the opportunity to observe the material from which some of these conclusions were drawn, and it is my firm opinion that only by classifying the work done can we come to any definite conclusion as to what to do in these forms of tuberculosis.

Every year, many hundreds, perhaps thousands of patients having pulmonary tuberculosis are given the advantages of surgical therapy yet these represent only a small minority of those patients who might be treated with benefit. Records show that in the New England states only about 4.5 per cent. of the patients are given the benefit to be obtained by use of the various surgical methods and this is probably representative of the country at large. In the larger clinics of the country from fifteen to seventeen per cent. of the patients are treated by surgical methods and it is conservatively estimated that thirty per cent. of all patients with tuberculosis could be benefited by the application of some one of the surgical procedures.

A consideration of the procedures used in the treatment of tuberculosis in any of its forms shows that the success of the treatment depends to a large degree on the amount of rest given to the diseased part. With this thought foremost in mind, we turned our attention to the larynx to determine just how much comfort and relief could be given a patient with tuberculosis of the larynx by resorting to surgical measures.

Our problem as we found it was to relieve patients suffering from severe pains on swallowing, with its diminished food intake and the rapid failing of the patient.

Since the superior laryngeal nerve, essentially the sensory nerve of the larynx, was our chief offender, we, for a long period injected this nerve with alcohol, but we were met with only varying and temporary results and with occasional mild complications.

Feeling that we needed something that was absolutely safe and would give permanent results, we next began to section the superior laryngeal nerve and with the most gratifying results to all concerned. Inasmuch as our patients soon began to enjoy their nourishment so vitally necessary in their fight, we were made happy again because of the comfort and rest afforded to these ever hopeful individuals who are always looking to us for their continued existence.

Dr. Francis Lederer, Chicago: We divided up our discussion because of the brief time allotted our presentation, so that I think it might be well to summarize some of the material that appears in this paper. After all, the therapy of 1934 is not so materially different from that of the 80's. However, we are struck by the occasional discussions which appear in the literature, because of the hit or miss attitude of the therapy, the same attitude persisting in the minds of most laryngologists on the entire subject of the therapy of laryngeal tuberculosis. Time after time you see in the literature an article advocating a certain agent in the treatment of laryngeal tuberculosis, without having any definite object in mind in selecting a certain type of case for that particular remedy. In other words, you as the reader have the idea that all laryngeal tuberculosis fits in with that therapy; then you are chagrined when you institute such therapy and the results are not forthcoming as the author indicated, with drug therapy or light therapy. Outstanding in this respect are such things as voice rest. We find a recent author referring to voice rest as a dreadful thing. It is certainly dreadful to institute voice rest where there is an ulcerating lesion in the larynx, remote from cords or arytenoids. We would speak in the same manner of quartz light therapy. The minute you mention laryngeal tuberculosis to the average physician, the quartz lamp is rushed into the sickroom. It is the main part of the therapy in most minds. In the detailed paper we did indicate the quartz or other heliotherapy but for only certain selected types of cases. No internist who is acquainted with pulmonary tuberculosis immediately rushes his patient into the sunlight—it might cause the process to break down more rapidly than otherwise. These are only a few of the examples of how we try to rationalize our therapy along sane lines.

I was struck by a question that Chevelier Jackson placed before us in which he asked for a rational therapy, for a patient with laryngeal stenosis, the patient still having an inactive focus in the lung, or maybe a latent process. That latency is a very difficult thing to evaluate. One never knows when a lesion is completely quiescent or completely arrested or cured, so if you institute a certain policy in the presence of an active focus, the individual having a positive sputum for instance, that is not in line with our suggested therapy. These slides, I know, illustrate the manner of our ap-

proach to the problem at hand. Our first consideration is the prophylaxis in all cases of pulmonary tuberculosis, the second, is the objective of the active treatment, relative to nutrition and the preservation of laryngeal function and the third is the evaluation of all the agents such as drugs with their various means of application, heliotherapy, cauterization and the many surgical procedures which are practiced and advocated.

Dr. Joseph Beck, Chicago: I think you will all agree with me that this presentation was complete, as complete as it could possibly be within the time limit, and my comment is this: That men in institutions who are treating tuberculosis in general should have associated with them not only a laryngologist but one who is well equipped to handle this situation. You saw this in the analysis of the work shown here today. Personally I have little to add from my experience. I feel if you are going to make any progress at all it will be by combination of medical and surgical confreres in institutions, and that treatment will only be rationalized in this way. You will find the literature flooded with this and that thing in treatment. This one scarifies the tuberculous process, rubs in dyes, gives a summary of so many cases improved, non-improved—but the basic information is not there as to what they are actually treating. I think this is an exceedingly refreshing summary of the work done. As I recall, in my early days of practice, tuberculosis of the larynx meant the end of the patient. There was nothing to be done.

Another thing is the matter of early interference, not waiting until the patient is starved and then do a gastrostomy; when he is suffering from pain and is exhausted for lack of air and then do a tracheotomy. This does not refer to the submucous type of tuberculosis of the larynx as shown by Mullen. There is no indication for surgical interference in such cases, as the patients seldom have pain of that kind or obstruction to breathing.

Dr. L. Z. Fishman, Chicago: I would like to add a small though important detail to the technic so well described by Drs. Savitt and Soboroff. There is one step in the procedure of this operation in the human which has always made the exposure of the internal branch of the superior laryngeal nerve relatively more simple than otherwise. As we know, this branch courses downwards within the fascia of the thyrohyoid membrane at an angle of about 45 degrees in relation to the anterior border of the sternocleidomastoid muscle. If the blades of a scissors are inserted into and opened in this fascia, somewhat anterior to the lateral thyrohyoid ligament but in a position that is parallel to the course of the nerve as designated by the 45 degree angle, the nerve will usually present itself as a white band, well-defined from its surrounding areolar connective tissue.

Dr. Simon H. Soboroff, Chicago: I wish to thank the gentlemen who were so kind with the discussion. In my nervous haste to finish reading the paper, I believe I neglected to say that the work was done in the Municipal Tuberculosis Sanitarium, and it was largely through the kindness of the chief of this department, Dr. Lederer, that we were able to do the work. Dr. Fishman's suggestion will be tried out in the next series of cases. It is the little things that must be watched, the little cough, the slight hoarseness, the slight sore throat. We have had that experience at the sanitarium. The patient has a complaint of hoarseness of about a month. Examination of the larynx shows extreme infiltration. When asked if he has been hoarse before the patient says yes, that he went to a doctor and was given some cough medicine. It is important to examine the larynx and notice these things in time.

RATIONALIZATION IN THERAPY OF LARYNGEAL TUBERCULOSIS — THE EVALUATION OF THE LARYNGEAL FOCUS IN PULMONARY TUBERCULOSIS

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Is laryngology arriving at or is it passing the stage of adolescence? Or is it conceivable that it has become quite mature and sedate as a specialized science of the art of medicine in contrast to its status during the period of 460 B. C., when the larynx was considered as connecting the urinary bladder with the pharynx? Perhaps we are certain today, thanks to Galen's work in the second century, that the heart is not the organ of voice. Considering all things relatively, we are not so certain that laryngology, particularly the subject of laryngeal tuberculosis, is as familiar to many of us as it is entitled to be. This statement regarding the subject under discussion is not made on the basis of fancy, but upon that of the palpable lack of available information which is shown by present-day publications concerning tuberculous disease of the larynx, particularly its therapy.

It has always appeared to us that there has been a laxity in the treatment of this affection, first through the lack of understanding of the disease, and second because of a phlegmatic or pessimistic attitude in regard to this complication. The fatalistic attitude of many physicians of past decades regarding the incurability of laryngeal tuberculosis, is not as prevalent as

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formerly, although there are still those who could be living in that period, preceding 1880, before the advent of rational therapy of the larynx. Associated with this early advance, we recognize the names of Albers (1829) who advocated the performance of tracheotomy for the rest of the larynx; Trousseau and Belloc (1837) who advised steam inhalations, powders, etc.; Horace Green (1838) who practiced the topical application of silver nitrate to the larynx; Moritz Schmidt (1880) who recommended such procedures as tracheotomy and scarification; Krause (1885) who indicated lactic acid as a therapeutic aid; and Heryng (1886) who advised curettage.

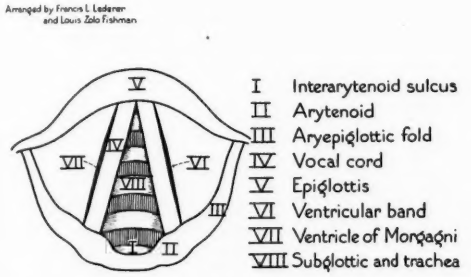

Fig. 1. Stress is placed upon the origin of laryngeal pathology from a pulmonary tuberculosis, attaching equal importance upon concise clinical and histopathological interpretations and localization of the laryngeal lesion. These three factors form the basis of an equilateral triangle for comprehensive diagnosis and the foundation for rational therapy. The ultimate goal is therefore depicted at the apex which is arrived at by means of the basic steps.

Since that time there have been many other therapeutic measures advised, some of which have been continued or improved upon, and many others which have been discarded. The treatment during all of these years has varied from one extreme of passive and hopeless expectancy to the other of energetic surgical interference. Many of the methods which have been recommended are successful in some hands, but have failed in others because of improper technique and the lack of individualization regarding the choice of cases. The ease with which these errors are committed may be demonstrated in a vivid manner by pointing only to the wide variations in certain conceptions regarding this disease. For example, specific involvement of the larynx in pulmonary cases varies in occurrence from 3 to 97 per cent. in the statistics according to their source; in the average variety of cases, 3 to 25 per cent. would more nearly approach a correct statistical figure. In our own institutional experience we find that 15 per cent. of patients with pulmonary tuberculosis have laryngeal tuberculosis. To treat all cases regardless of lung pathology and type of laryngeal involvement by universally applying one form of therapy is obviously unfair, even in the light of good results. Reports are being published recommending one form of therapy or another as a routine procedure in all cases of laryngeal tuberculosis. This lack of discrimination we look upon as being illogical. We consider it to be of greatest importance, in reporting a type of therapy, that we indicate in no uncertain terms the method according to its separate and individual indications.

Although we intend to lay greater stress upon the local therapy of the larynx in our presentation, it is essential that tuberculosis of the larynx be defined in a general introduction to the subject, as a disease which is always secondary to a pulmonary lesion; so far, no case of unquestionable primary laryngeal tuberculosis appears among our own cases. Also, very little controversy exists as to the relation between the course of the pulmonary and the laryngeal pathology; the one may increase in severity while the other becomes arrested or cured, although the frequency of laryngeal involvement undoubtedly increases in the terminal stages and in the severe types of pulmonary tuberculosis. Nor can we stress sufficiently the fact that tuberculosis of the larynx is not indicative of a terminal stage of pulmonary tuberculosis, but may be the first sign of a hidden pulmonary focus. This fact is demonstrated not uncommonly when we point to the presence of pulmonary tuberculosis upon the basis of our diagnosis of a tuberculous lesion within the larynx, and must subsequently force the internist, before he succeeds in confirming our interpretation, to persist in his search for an earlier or preceding focus.

In the same manner in which we present first the problem of the focus within the lung, so do we consider first at the patient's bedside, before therapy of the laryngeal disease is rationalized, the degree of pulmonary pathology present. It is obvious that a hopeless case of far-advanced pulmonary infection and generalized debility is also hopeless from the specialized point of view.

We may only do those things which will afford the patient as much comfort as possible, because such a patient shows that he has neither the resistance to overcome the bacillary invader, nor the ability to satisfy any other reparative demands from other regions of the body. Surgical procedures are, then, definitely contraindicated for this patient, except as a last resort. These exceptions are vital and occur when a secondary lesion is of such a nature,—even if it is within the larynx,—that it must in itself lead inevitably to death. There are various methods for ascertaining and indicating to the physician the status of the pulmonary and general condition of the patient, such as pyrexia, tubercle bacilli in the sputum, loss or gain in weight, shifts in the blood picture and lastly, blood sedimentation. Briefly, these findings form our basis for indicating or contraindicating certain types of caustic or surgical therapy. The problem is an extensive one and must be interpreted with the cooperation of the internist.

The second group of considerations is our own evaluation of the local disease. We have divided the basis for such an evaluation into two major classes according to: 1. the pathologic character of the laryngeal lesion, which establishes therapy on the basis of both the gross and microscopic pathology and the corresponding indications of the clinical course (progressive; arrested or latent; or cured), and 2. its anatomic location, which establishes therapy on the basis of both the histologic structure and physiologic function. A qualitative diagnosis, embodying such descriptive characteristics as these, permits universal terminology, and, in the final analysis, universal therapy locally; logical sequence in therapy follows. "Quality diagnosis" expresses our desire to impress upon the physician that an understanding is absolutely necessary of the various general forms and manifestations of the disease correlated with definite local effects of laryngeal tuberculosis. To accomplish such comprehensive information from our diagnosis, we must recognize pathologic types, anatomic localizations and clinical forms (Figure 1). Regarding the pathologic character of the lesion, the same basic processes and resulting objective symptoms pertain to the larynx histologically as elsewhere in cases in which mucous membranes, muscles, cartilages, bones, or joints are attacked by the tubercle bacillus. While laryngeal lesions have a basic histopathologic character, they are subject to greater variations first, because of the specialized anatomy and functional activity of the larynx, and second, because of the presence or absence of secondary infection. Objectively, we recognize in a simple manner early stages of invasion by the appearance of hyperemia, followed by infiltration (productive or non-productive), then ulceration (superficial or deep; active or sluggish) followed by the appearance of granulation tissue (tuberculoma or papilloma); perichon-

Fig. 2. The order of frequency in which various parts of the larynx are affected in laryngeal tuberculosis. Precise localization of the laryngeal lesion anatomically is emphasized because of consequent dysfunction of local physiological acts and general ill-effects upon the patient. The areas may be involved singly or multiply. Sites of predilection vary according to institutional experiences.

dritis or chondritis and possibly necrosis with abscess formation, periostitis and even osteomyelitis. Such a chronologic course may be termed clinically as "progressive" (Figure 2). Combinations of these various pathologic processes may be coexistent in the larynx of a patient. Furthermore, one type of lesion may be progressive while another may become arrested (latent) or cured. If the cricoarytenoid or cricothyroid articulations become involved, any one of the types of tuberculous arthritis results, which may lead eventually to ankylosis of the joint and fixation of the vocal cord. If any of the cartilages are attacked, the symptom complex of laryngeal perichondritis presents itself. Again, reactions to therapy and disease differ according to whether the cartilage is hyaline or fibroelastic,

and according to whether the type of epithelium is non-ciliated stratified squamous, ciliated pseudostratified or ciliated columnar. Lastly the use of the anatomic term in the diagnosis to designate the location of the laryngeal pathology will at once indicate what the physiologic prognosis may be and what therapeutics are contraindicated to safeguard, if possible, the patency and the phonatory movements of the larynx.

Rational Therapy Based Upon Pathologico-Clinic and Histologico-Anatomic Characteristics of the Tuberculous Lesion with the Larynx. Because this form of laryngeal disease occurs more frequently than any other specific disease of the larynx, we have always emphasized laryngeal prophylaxis in the routine management of the tuberculous.

PROPHYLAXIS IN THE TUBERCULOUS

1. A high standard of the patient's physical and mental status must be maintained.
2. The larynx must be examined frequently in all cases of tuberculosis as much depends upon early detection of local pathologic processes. Attention must be directed to the minor laryngeal complaints before they attain major proportions.
3. Local areas, when non-tuberculous, must be treated to prevent the resistance of the parts from becoming lowered. Chronic laryngitis must be treated as well as abrasions and superficial ulcerations.
4. Voice hygiene or absolute vocal rest is important. Proper use of the voice is to be emphasized, especially for those who are subjected to coughing paroxysms which alone punish sufficiently the vital components of the larynx.
5. The so-called "common cold" particularly should be watched and properly treated.

Evaluations of the disease of the larynx on the basis of the foregoing facts enable us to speak about lesions, wherever they may occur, in vivid terms, so that in a great measure, we may subscribe to rational diagnosis and treatment. We briefly present our qualitative concepts of the main pathologic groups and active therapeutic principles involved in each of these:

OBJECTIVE OF THE ACTIVE TREATMENT

1. To relieve symptoms which not only interfere with comfort, but defeat one of the main objectives in the general treatment, viz., nutrition.
2. To obtain complete arrest of the tuberculous infection, pulmonary and laryngeal.
3. To cure it in the larynx, preserving, if possible, laryngeal function.

Vascular Changes: Hyperemia, generally, is not a serious laryngeal complication of pulmonary tuberculosis, and calls for mild forms of therapy such as sunlight or mercury vapor irradiations. These must be applied directly to the inner surfaces of the larynx if therapeutic results are to be expected. In the very early stages of laryngeal tuberculosis we always place the organ at complete rest and feel that even heliotherapy may activate the local process. Roentgen ray therapy has had enthusiastic claims made for it and excellent results have been reported following its use in incipient cases. It may be stated here as applying to all pathologic types of lesions that immobilization of the larynx obtained by complete silence,—which does not mean that whispering may take the place of complete silence,—is indicated only when the vocal cords or the cricoarytenoid articulations are involved. Since the most common site for the tuberculous lesion is the interarytenoid sulcus above the level of the vocal cords (supraglottic), voice rest is of value in the greater number of early cases of tuberculous laryngitis. Incidentally, hoarseness or aphonia is also, on the same basis, an uncommon or late symptom. One or the other may be *present at any time during the course of* pulmonary or laryngeal tuberculosis for other reasons, such as a non-specific laryngitis or phonasthenia. Hyperemia of the interarytenoid sulcus may lead to a diagnosis of the pathogenesis of *spasmodic coughs or odynophagia in these patients*; in such an event, mild astringents may be prescribed either in the form of topical applications or steam inhalations. Indiscriminate swabbing is not advised because it is uncomfortable, irritating and ofttimes injurious.

Anemia, when local, indicates an infiltration which must be differentiated from pachydermic states. When generalized in the larynx and pharynx, it will be found that it is merely another mucous membrane symptom of secondary anemia or debilitation and by no means does it indicate local tuberculosis. In the latter instance, therapeutic measures are obviously to be directed against the pulmonary disease and general debility. The pallor which is considered to be associated with laryngeal tuberculosis is one that is combined with an infiltrative edema producing a picture characteristically ashen grey in appearance, and entirely different from that seen in states of secondary anemia.

Infiltration is pathognomonic when it involves the interarytenoid sulcus, usually on one side more than the other, and when non-specific

pachydermia laryngis can be eliminated from the clinical diagnosis. The next sites of predilection for infiltrative processes are the arytenoids and aryepiglottic folds. Next in frequency of occurrence, are the true cords, false cords, the epiglottis and, lastly, the sinus Morgagni, subglottic area, and trachea. In infiltrative and mildly ulcerative cases, definite influence and even an arrest of the process may be brought about by heliotherapy. In advanced types the beneficial effects of such agents are questionable. The galvanocautery offers the best type of therapeutic attack against such infiltrations, because, by means of igni puncture or even mass cauterization, these lesions may be destroyed. The consequent fibroblastic reaction is particularly excellent and serves to encapsulate further in dense scars, active foci which may have been missed and which are adjacent to cauterized areas. On the basis of the histologic character and established reactions to trauma of the cricoid, arytenoid and thyroid cartilages, galvanocautery is never applied to these cartilages. The epiglottis, on the contrary, because of its fibroelastic structure and firmly adherent perichondrium, may be cauterized whenever necessary. Care must always be exercised to avoid the superior laryngeal arteries in any traumatic procedure on the epiglottis. Again, if an infiltrative process involves the anterior commissure, the anatomic location contraindicates cauterization or any form of surgical procedure because of the danger of scar formation which may result in glottic stenosis. Roentgen therapy, tuberculin injections or other less established methods may be attempted as secondary therapeutic choices in these cases. Silence is a good form of therapy when the true cords, membranous or cartilaginous, are infiltrated.

Ulcerations, are differentiated according to their appearance in diffuse or in asymmetrical groups. The diffuse process occurs in the larynx as part of the lesions of miliary tuberculosis. Such ulcers develop too early following the formation of miliary tubercles for these tubercles to be noted clinically. The asymmetrical groups are essentially limited as local processes. Therapy for acute miliary tuberculosis of the larynx is as hopeless as that which may apply to the generalized miliary characteristics of this fulminating and disseminating form of pulmonary infection. Such is not true of the more localized ulcerations. If we continue according to the principles of rationalization in therapy, it is evident that chronic, sluggish ulcers may respond to topical applications of neutral quinine hydrochloride, formol, lactic acid, paramonochlorophenol and trichloracetic acid. The galvanocautery is more effective for combating larger ulcerations, just as it is for destroying infiltrative lesions *per se.* The membranous portion of the true cords may be cauterized massively, yet the healing process will almost fill the deficiency with scar tissue so that even phonation may be restored eventually. When ulcerations appear, they induce pain; when they involve cartilages, the pain accompanying the perichondritic or chondritic process is excruciating. In addition to immobilizing the larynx by silence, it is often necessary to inject the sensory (internal) branches of the superior laryngeal nerves. This is performed first on the side which appears to be causing the greatest amount of pain; if the relief obtained is not sufficient, the nerve of the other side is injected about five days later. A few instances of positive relief will be impressive and will illustrate the importance of so simple a procedure. In the event that these measures do not relieve the pain entirely, section of the superior laryngeal nerves themselves or their sensory branches is resorted to; anomalous branches may defeat the purpose of the operation upon the sensory branches, and glossopharyngeal neuritis may defeat the procedure upon the superior laryngeal nerve. Finally, gastrostomy is a rational procedure when it is desired to remove all sources of irritation to the ulcerated parts, even that trauma which is produced by the passage of liquids and the attending movements of the larynx during deglutition. There remains for our consideration, in the discussion of the treatment of perichondritis, lesions of this character involving the epiglottis. In this instance, if the upper third is diseased, partial epiglottidectomy offers an effective and thorough method for eradicating the local disease, the dysphagia, the respiratory interference, or the aspiration of food and drink. Small ulcers of the epiglottis may be treated in the same manner as suggested for similar lesions in other parts of the larynx.

Tuberculoma is a specific type of infiltrative reaction of the tissues to tuberculous disease of the larynx consisting mainly of connective tissue, resembling grossly a fibroma or papilloma, or it may be a typical tuberculous granuloma. Tumor

formations of tuberculomas occur most frequently as solitary lesions, although they may present bizarre forms of extensive excrescences. This type of lesion is most commonly found upon the posterior wall, in the interarytenoid areas, the ventricle of Morgagni and the anterior commissure. In rarer instances, they are found on the vocal cords, epiglottis and ventricular bands. In the ventricle of Morgagni, tuberculomas may simulate a state of ventricular prolapse. Tuberculomas may be removed surgically, followed by galvanocauterization of their bases.

Granulations are usually found overlying ulcerations, but may assume a papillomatous appearance, masking the original pathology. The treatment is essentially like that employed for ulcerations, excepting that it should be more vigorous. The diseased areas may be removed surgically when the risk is good, and when not good, performed as a last resort.

Reparative processes, as well as progressive ones, require rational therapy. By this statement it is meant that the ability of the laryngologist to abstain from interference in certain latent or arrested processes is as vital therapeutically as his foresight in indicating and skill in performing laryngeal operations. The clinical differences between latent and cured lesions are of no value, since the terms are employed merely on a presumptive basis of inactivity. A latent or arrested lesion is considered as cured after it has remained stationary for a period of years. The fact that tuberculous foci may be activated particularly by trauma, even after a period of latency extending over 10, 15, 20 or more years, establishes the danger of attempting plastic procedures within the larynx in the presence of arrested tuberculous processes. There is, as may be deduced, a great difficulty in the distinction between permanent cure and latent, quiescent or arrested states.

OUTLINE OF ACTIVE TREATMENT

1. *Drugs.*
   1. Intralaryngeal Instillations or Sprays.
      Chaulmoogra oil, Mentholated oils, Orthoform emulsion, Cocaine, Butyn.
   2. Topical Applications.
      Formalin, Lactic acid.
   3. Insufflation of Powder.
      Orthoform, Thioform, Methylviolet, Iodoform, Anesthesine, Zinc stearate.
   4. Inhalation of Vapors.
      Benzoin, Menthol, Creosote, Guaiacol.
   5. Lozenges.
      Orthoform, Anesthesine.
2. *Heliotherapy and Radiotherapy.*
   1. Finsen light (carbon arc).
   2. Direct reflected sunlight.
   3. Ultraviolet.
   4. Roentgen ray.
   5. Radium.
3. *Cautery.*
   1. Thermo or Electrocautery.
      Galvanocautery, Surgical diathermia.
   2. Chemical Cautery.
      Trichloracetic acid, Neutral quinine hydrochloride, Lactic acid, Formol, Paramonochlorophenol, Formaldehyde, and Silver nitrate.
4. *Surgical.*
   1. Endolaryngeal and extralaryngeal procedures.
      Currettage and Scarification, Tracheotomy, Epiglottidectomy, Superior laryngeal nerve-blocking and resection, Thyrotomy, Laryngotomy, Hemilaryngectomy, Laryngectomy.
   2. General Surgical Procedures.
      Collapse therapy, Resection of phrenic nerve, Extrapleural thorocoplasty, Gastrostomy, etc.

One cannot conclude a discussion of laryngeal tuberculosis without a word about the manner in which such cases are best controlled, i. e., office or home management, vs. hospital or sanitoria isolation. The careful and frequent check on all pulmonary cases requires routine laryngoscopic observation which, in itself, does not necessarily constitute an institutional problem. Despite the present tendency to the contrary, it can be justly stated that therapy can best be carried out in institutions under close supervision and specialistic observation. Laryngeal tuberculosis indicates a highly infectious condition which calls for isolation and the strictest and most diligent discipline of the patient. Such a regime is best accomplished in institutions where conditions are inherently adaptable to prophylactic and active therapeutics. It is relatively easier for an institutionalized patient to acquire the desire of cooperating with the physician and to learn stoicism than it is for a patient who remains at home where all of his own problems and, in addition, those of his family are constantly brought to his attention. There is no need to discuss the merits of maintaining a healthy mental as well as a healthy physical state to assist the convalescence of tuberculous patients particularly.

SUMMARY

1. Stress is placed first on the origin of this specific form of laryngeal pathology from a pri-

mary pulmonary tuberculosis, it being recalled, however, that laryngeal complications, when they occur in these tuberculous patients, are not necessarily specific in character; equal importance is attached to concise clinical and histopathological interpretations of the pulmonary and laryngeal states; finally, precise localization of the laryngeal lesion anatomically is emphasized with reference to consequent dysfunction of local physiological acts and subsequent local and general ill-effects on the patient.

2. These three groups of factors are discussed as prerequisites to the formation of a basic equilateral triangle for comprehensive diagnosis; it follows that only upon such a complete and logical foundation can the multitudinous types of therapy be pyramided toward success in an application to the almost equally great number of tuberculous manifestations within the laryngeal structure. The conclusion becomes obvious, "that any departure from the above principles may be stigmatized as empirical if not entirely unsound."

3. Tuberculosis of the larynx is similar in its pathology to tuberculosis in other organs, being modified however, by the anatomy of the larynx and its functional activities. The pathology of the lesion indicates the clinical course of the disease and the type of therapy. The anatomic location of the lesion indicates the physiologic prognosis of the organ and the type of therapy which will safeguard, if possible, the functions of the larynx; it also permits a knowledge of the response to various types of therapy on the basis of the histologic structure of the part involved.

4. Immobilization of the larynx entails complete silence and prevention of deglutition. Complete silence alone is indicated only when the vocal cords or when the cricoarytenoid articulations are involved; extensive perichondritic lesions call for more radical measures, such as tracheotomy and gastrostomy, to insure greater degrees of immobilization of the larynx than is obtained by silence.

5. The relative merits of the various drugs are not easily determined. Much of the success of any of the therapeutic measures is dependent upon the interest and diligence of the physician in charge and upon the status of the pulmonary condition. The matter of a careful technique and the choice of the proper indication stands paramount.

6. The epiglottis has no function in phonation or deglutition, nor is its removal mutilating to the larynx; partial epiglottidectomy is vital when extensive infiltration or ulceration of the epiglottis results in aspiration of food, excruciating pain, or dysphagia.

7. Injection and section of sensory nerves are palliative and not curative procedures *per se*, except when such procedures permit rest and intake of nourishment.

8. Laryngeal tuberculosis may be arrested or cured without extension, no impairment of the physiological functions of the larynx need result, though defects may remain.

9. Institutionalization, for isolation, discipline and control of the patient is recommended.

10. The maxim is offered, "It is better to have a live and well patient without an intact larynx and voice than to watch his exitus as the result of pain or starvation; it is also to be desired that a cured patient retain as much of his larynx and voice as is compatible with rational therapy."

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NUTRITION WORK IN THE SPRINGFIELD, ILLINOIS, SCHOOLS

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During the past two years it has been deemed advisable to give considerable attention to the nutrition of children attending school. This was done to avoid the penalty of lowered resistance, sickness and other consequences resulting from undernourishment, following in the wake of inadequate food supplies experienced by many families on account of the economic depression.

Method of Procedure. At the beginning of each semester all the children in the elementary schools are measured, weighed and listed on Classroom Weight Record Sheets as shown in chart 1. In the public schools this is done by the Department of Physical Education. In the parochial schools the school nurses do this work.

The percentage variation from the average normal weight for age and height, as given in the

Read before Section on Public Health at annual meeting of Illinois State Medical Society at Springfield, May 16, 1934.

CHART 1. INSTRUCTIONS FOR WEIGHING AND MEASURING

Room..... Grade..... School..... Teacher's Name..... Year.....

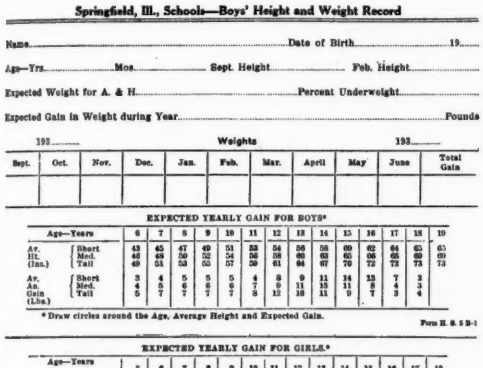
Names	Sept. Age	Ht.	Wt.	Norm. Wt.	+ —	% U. Wt.	Feb. Age	Ht.	Wt.	Norm. Wt.	+ —	% U. Wt.

(1) Have teachers fill in blanks below and give names and ages of pupils in Years and Months, making separate sheets for Boys and Girls. (2) These sheets are to be sent with pupils when coming to be weighed.

WEIGHING: (1) Have children remove shoes, coats, and sweaters. (2) Have child stand with toes on front edge of scales platform, hands down to side and head and shoulders up. (3) If possible, have two people help, one for measuring and the other for weighing. (4) The plan for weighing in each school is to be worked out with the principal and the person or persons who are to be responsible for this work in the respective schools.

Baldwin-Wood Tables, is figured and entered on the Classroom Weight Records.

An Individual Weight Record Card as shown in chart 2, is started for each child found ten



Springfield, Ill., Schools—Boys' Height and Weight Record

Name..... Date of Birth..... 19.....

Age—Yrs..... Mos..... Sept. Height..... Feb. Height.....

Expected Weight for A. & H..... Percent Underweight.....

Expected Gain in Weight during Year..... Pounds

193..... Weights 193.....

Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	April	May	June	Total Gain

EXPECTED YEARLY GAIN FOR BOYS*

Age—Years		6	7	8	9	10	11	12	13	14	15	16	17	18	19
Av. Ht. (Ins.)	Short	43	45	47	49	51	53	54	56	58	60	62	64	65	65
	Med.	46	48	50	52	54	56	58	60	63	65	68	69	69	69
	Tall	49	51	53	55	57	59	61	64	67	70	72	73	73	73
Av. An. Gain (Lbs.)	Short	3	4	5	5	5	4	8	9	11	14	13	7	3	
	Med.	4	5	6	6	6	7	9	11	13	11	9	4	3	
	Tall	5	7	7	7	7	8	12	16	15	9	7	3	4	

* Draw circles around the Age, Average Height and Expected Gain.

Form H. S. S B-1

EXPECTED YEARLY GAIN FOR GIRLS.*

Age—Years		5	6	7	8	9	10	11	12	13	14	15	16	17	18
Av. Ht. (Ins.)	Short		43	45	47	49	50	52	54	57	59	60	61	61	61
	Med.		45	47	49	52	54	56	59	62	63	64	65	64	64
	Tall		47	50	53	55	57	59	62	64	66	67	67	67	67
Av. An. Gain (Lbs.)	Short		4	4	4	5	6	6	10	13	10	7	3	1	
	Med.		5	5	5	6	8	10	13	10	8	4	3	1	
	Tall		6	6	6	9	11	13	9	8	4	1	1	1	

* Draw circles around the Age, Average Height and Expected Gain.

Form H. S. S G-1

Chart 2. (Above) Reduced from boy's 6x4 blue card. (Below) Part of girl's pink card. Reverse side of cards is a form for recording data for several terms of school.

per cent. or more underweight. The expected gain in weight for the ensuing year is determined for each such child and entered on this card.

All the children found ten per cent. or more underweight are re-weighed each month, and the results entered on the individual records. Whenever a child shows no gain at such weighings, an inquiry is made in regard to attacks of acute illness and where these have occurred the entries are "circled."

Similar individual records are kept on children in contact with a case of tuberculosis and on all pupils in special classes, such as the Sunshine School, Crippled Children's School and Sight Saving Class.

An Individual Monthly Weight Record is also kept for all children who were less than ten per cent. underweight when first weighed and who made no gain during the following semester.

Accuracy and Uniformity. In order to make the results in the various schools and the different weighings comparative, it is necessary that a uniform method of weighing, measuring, computing and classifying the results be employed.

Turner,[1] in discussing the Precision and Reliability of Underweight Measurement, showed the results of using various methods in computing the per cent. of children ten per cent. underweight in a group of 475 children. With variations in the method of procedure and computation, the percentage of underweight children ranged from 4.8 to 25.3 per cent. with the five different methods employed.

In order to prevent such variations in the results, definite written instructions were issued and followed in determining and computing the weight variations of the pupils observed.

The following are some of the pertinent points covered by these instructions:

All scales are to be inspected and sealed by the City Inspector of Weights and Measures.

Children are to be weighed with their outdoor clothes, pocket contents and shoes removed. Results are recorded according to the nearest numeral.

Age used for computation shall be that of the nearest birthday.

Monthly weighings are made on or before the 10th day of each month.

Compare last and first weights of period.

Results of Initial Weighing. The results of the weighing of all the children in the elementary schools, exclusive of those in special classes, immediately after the reconvening of school in September, are considered as the initial weighing.

The results of these weighings for the years 1932-33 are shown in Table 1.

TABLE 1

Number and Per Cent. of Children Ten Per Cent. or More Underweight in the Springfield, Illinois, Elementary Schools at First Weighings—1932-1933

1932			1933		
	Ten Per Cent. or More Underweight			Ten Per Cent. or More Underweight	
Number Weighed	No.	%	Number Weighed	No.	%
10,719	1,427	13	11,170	2,399	21

1. Turner, Clair Elsmere. Amer. J. of P. H., Sept., 1929, pages 969-77.

These weighings showed 21 per cent. of the pupils, 10 per cent. or more underweight in the fall of 1933, as compared with 13 per cent. in the preceding fall.

This represents an increase of 62 per cent. in 1933 over 1932. This percentage increase ran fairly uniform through the public and parochial school systems.

In September, 1932, the proportion of underweight children in the various schools ranged from 6 to 19 per cent.

As a general rule, the highest percentages of underweight children were found in the schools of the poorer districts of the city. The highest per cent. was found in the Central district, where many residences have been converted into rooming houses and small apartments, and where many mothers are employed.

In September, 1933, the results in the individual schools varied from 8 to 42 per cent. and the highest percentages were found in the districts where the largest proportion of families were on relief.

Weighings at End of First and Second Semesters, School Years 1932-33. During the first semester of 1932, 1,379 of the children found 10 per cent. or more underweight in September were kept under observation and weighed monthly. At the end of the semester 27 per cent. showed no gains in weight. Physical examinations were made of nearly all the children who failed to gain in weight. In approximately 80 per cent. nothing was found which might be considered as causing or contributing to the malnutrition. In the other 20 per cent. the most frequent findings were adenoids, enlarged tonsils, and inflamed cervical lymphatic nodes.

At the end of the second semester of the school year 1932-33, 25 per cent. of the 1,502 kept under observation showed no gain in weight. These represented 3.4 per cent. of the children enrolled in the elementary schools.

At the beginning of this semester the children who were less than 10 per cent. underweight in September, and made no gain during the first semester, were taken on for observation and monthly weighing. These, combined with the children found 10 per cent. or more underweight, represented 13.2 per cent. of the entire enrollment.

It is evident now that the failure of approximately 25 per cent. of the underweight children to gain in weight during the school year 1932-33 was a forerunner of what occurred in the following summer, for when the children were reweighed in the fall of 1933, it was found, as referred to above, that the percentage of children 10 per cent. or more underweight had more than doubled.

Milk Fund Established and Milk Feeding Stressed. When the great increase in the percentage of underweight children in the fall of 1933 was first definitely noted, it was decided that as an emergency measure every child found suffering from malnutrition should be supplied with milk in school.

A physical inspection of 1,493 underweight children showed distinct evidences of malnutrition in 63 per cent. Markedly enlarged tonsils or adenoids were found in 18 per cent. of all the children examined.

Following this examination and classification, a letter was sent to the parents of all the children in the public elementary schools, found undernourished, advising that the child be provided with two half pints of milk in school. Parents were also asked to indicate if they could supply this milk at their own expense, in case they desired their child to be provided with milk, as suggested.

In the parochial schools the importance of providing the undernourished children with milk was stressed in personal contacts with the teachers, parents or older children of the family.

The tabulation of the replies received from the parents showed that in slightly over a third of the cases the parents indicated that they were financially unable to supply the milk in school as recommended.

The Council of Social Agencies was appealed to for a Milk Fund to supply milk to the undernourished children in these needy families. After a careful review of the evidence, the Council agreed to raise such a fund, and in anticipation of the Drive for a Community Fund, which was planned for the near future, agreed to start forthwith to provide the milk in the public schools for these children. The parochial schools arranged to handle the problem in the various parishes where it was found that aid was needed.

Naturally, the drives to raise the general and local milk funds resulted in a lot of publicity in

regard to the necessity of milk for undernourished children. This, together with the home visiting, done by the social agencies to check on the use of milk in the home by families on relief, in which undernourished children were found, helped to arouse the interest of the parents in the proper feeding of their children.

Although all these children were at first supplied with milk in school—either one or two pints—depending on their degree of malnutrition, the aim was to supply the necessary amount of milk in the home. When this was accomplished in a given case, the supplying of milk in school, for children in this family, was discontinued.

All the children for whom free milk was recommended in the public schools were thus supplied with milk or oatmeal in the schools early in the Fall of 1933. Children with marked evidences of malnutrition were given a half pint in the morning and afternoon. The others received a half pint in the middle of the forenoon. All the families from whence these undernourished children came were investigated by the social agencies or nurses and when reported as receiving an adequate supply of milk and other essential foods in the home, were dropped from the school lists. In May, about 72 per cent. had been dropped.

Approximately 50 per cent. of the children in families not on relief, or not requesting financial aid for supplying milk to their undernourished children in school, were provided with milk in the schools at the parents' expense.

Thus approximately 75 per cent. of all the under-nourished children in the public schools were provided with milk in the schools, early in the Fall of 1933, soon after their condition had been definitely determined.

The principal accomplishment of this drive was that all the undernourished children in families on relief were supplied with milk in school at once, and with an adequate supply in the home, as soon as this could be accomplished. In the families on cash relief, this meant educating the parents in regard to the necessity of milk and other essential foods for the growing children.

The monthly re-weighings of all the children ten per cent. or more underweight afforded an opportunity for the nurse to contact these children individually and interest them in their diets and other measures necessary to improve their nutrition.

All of this had the salutary effect of interesting the parents in the nutrition of their children.

Results of Nutrition Work in the First Four Months of the School Year 1933-34. The results of the re-weighing in January, 1934, of all the children in the elementary schools who were ten per cent. or more underweight when weighed during the preceding September, are shown in Table 2.

TABLE 2

Results of Reweighing Underwreight Children—All Elementary Schools—January, 1934

Comparisons with September, 1933

Number Under Observation	2,230
Made Expected Gain (One-third of Annual)	1,765
Made Some Gain	305
Remained Stationary	79
Lost in Weight	25
Absent or Transferred	56
Per Cent Made Expected Gain (One-third of Annual)	79
Per Cent Made No Gain	5

An analysis of the results in the various schools shows that the greatest percentages of expected gains occurred, with a few exceptions, in the poorer residential neighborhoods. In these schools the percentages of children making their expected gains averaged 83 per cent. as compared with 70 per cent. in the better residential neighborhoods.

In the Catholic parochial schools 81 per cent. of the underweight children made their expected gains, as compared with 79 per cent. in the public and 77 per cent. in the Lutheran schools. The Catholic schools did not participate in the milk fund referred to before, but handled the problem of supplying milk and other essential foods to the needy undernourished children in their respective parishes.

In two public schools, 125 underweight children were given oatmeal and milk at the time of the morning recess. The children in these schools showed the same percentage of gain as the other schools in the poorer residential neighborhoods, namely, 83 per cent., yet these children were much more undernourished.

The outstanding fact disclosed by the re-weighing of the underweight children in January, 1934, was that only 5 per cent. failed to gain in weight since the preceding September (four months), as compared with 27 per cent. who failed to gain in the first semester (five months) of the school-year 1932-33. Employment under the CWA became quite generally effective in the early part of December, 1933, the

last month of the period, and therefore, could not have been the determining factor in bringing about the improvement in the nutrition of undernourished children, this last fall and early winter as compared with the results observed during substantially the same period of the preceding year.

Results of Experiment with a Noonday Health Class. In one school in the Central District where 19 per cent. of the children enrolled were found 10 per cent. or more underweight in September, 1932, the highest percentage found that year—and 30 per cent. were underweight when weighed in September, 1933, an experiment was started in the fall of 1933, with a view to giving these undernourished children a rest period in school in addition to supplementary feeding. This was deemed advisable because 51 per cent. of the underweight children in this school had shown no gain at the end of the school year 1932-33, as compared with an average of 25 per cent. in all the public elementary schools. This failure to gain occurred in spite of the fact that a special effort was made by the Parent-Teachers Association of that school to provide all the undernourished children with milk in school.

This is the school referred to above where a large percentage of mothers are employed and living in small apartments in made-over old residences.

It was noted that the children in these families were often expected to prepare their own noonday lunch.

It was, therefore, decided to have them bring their lunches to school and to supplement them with milk and one hot dish. The Parent-Teachers Association undertook the task of preparing the additional food and serving these children with their lunches thus supplemented, at tables set in the kindergarten room.

After eating their lunches, the children rested on cots for a period of 45 minutes under the supervision of a classroom teacher.

Twelve children in grades 4 to 8 inclusive continued in this so-called "Noonday Health Class" for a period of about five months. When the class was disbanded last March, all had made substantial gains, ranging from 5 to 13 pounds. Five made their expected annual gains in this period. All the others made more than half of their expected annual gains.

Although the experiment was conducted on a small scale, the results were encouraging especially in view of difficulty that had been experienced in the efforts made to improve the nutrition of these children during the preceding year.

Results of Weighing at End of Second Four Months Period School Year 1933-34. On account of the shortening of the current school year it was found convenient to summarize the results of the re-weighing of the underweight children at the end of periods of four months and to calculate the gains on the basis of a third of a year.

The results of the re-weighing in May, 1934, just completed, of the children in the elementary schools who were ten per cent. or more underweight when weighed in the preceding September are shown in Table 3.

TABLE 3

Results of Reweighing Underweight Children—All Elementary Schools——May, 1934

Comparisons with January, 1934

Number Under Observation	2,161
Made Expected Gain (One-third of Annual)	863
Made Some Gain	665
Remained Stationary	288
Lost in Weight	219
Absent or Transferred	126
Per Cent. Made Expected Gain (One-third of annual)	40
Per Cent. Made no Gain	23

An analysis of the results in the various school systems shows that the gains and losses ran rather uniformly throughout.

In the schools in the better residential neighborhoods 38 per cent. of the underweight children made their expected gains and 21 made no gains, as compared with a record of 41 per cent. making their expected gains and 25 per cent. making no gains in the other sections of the school district.

By comparing the results shown in Table 3 for the second four months, with those in Table 2 for the first four months of the school year 1933-34, it appears that the results in the latter period were far less satisfactory. In the second four months only 40 per cent. of the children under observation made their expected gains, as compared with 79 per cent. in the first period. Twenty-three per cent. made no gains in the second, as compared with five per cent. in the first.

Before accepting these figures at their face value, it must be determined if the results noted were influenced by other factors than the efforts made to improve the nutrition.

In this connection it must be noted that an

extensive measles epidemic occurred during the second four months period. A total of 1251 school children in the District had measles. This corresponds to 11 per cent. of the enrollment in the elementary schools.

The majority of the children who contracted measles and who were also under observation on account of their underweight showed losses, or no satisfactory gains in weight.

In addition the seasonal factor must be considered and is probably responsible for much of the difference observed in the results of the January and May weighings.

Mallinghansen's pioneer observations on the seasonal variation in the growth of children has been substantially confirmed by a host of investigators since his time (1883-86). He found that the seasonal variations in growth can be divided in three periods as follows:

1. Period of maximal growth—4½ months—begins in August and ends in mid-December.
2. Period of mean growth—4½ months—begins in mid-December and concludes at the end of April.
3. Period of minimal growth—3 months—from end of April to end of July.

Great differences in the rate of growth were found in these periods. The gain in weight in the maximal period often was two or three times as great as that of the middle period and all that was gained in the middle period might be lost in the period of minimal growth.

Roberts[2] reviewed the literature on this subject. The investigations quoted show that as a general rule "the period of maximum gain falls in the late summer and early fall; the period of minimum gain in the late spring and early summer, and that this tendency is more marked with girls than boys."

From these findings she concludes that "for a nutrition class to gain two or three times the average expected rate in the fall might mean no gain above what the children would have done without any nutritional care, since this is the period of rapid growth, whereas a gain of even this expected rate in the period of minimal growth might be real achievement." She recommends that expected gains should be computed according to the season of observation.

Tables showing the expected seasonal gains for various age periods of boys and girls are not readily available, but could be constructed from the findings of the various investigators in this field.

A comparison of the results shown in Table 3 with those in Table 4 throw light on this subject. In Table 4 the expected gains and losses are compiled on the basis of two-thirds of a year and the weights in May contrasted with the September weights.

TABLE 4

Results of Re-weighing Underweight Children—May, 1934—All Schools

May Results Compared with September, 1933

Total Number of Children Under Observation	2,164
Made Two-thirds of Expected Annual Gain	1,324
Per Cent. Made Two-thirds of Expected Annual Gain	61
Made No Gain	78
Per Cent. Made No Gain	4

By this comparison, the results appear to be much better due in part, no doubt, to the greater gains made naturally in the period of maximal growth from August to December. The percentage of children failing to make any gain is lower primarily because the period of observation is eight months instead of four.

If the extensive measles epidemic had not occurred in the second four months, the results shown by this comparison would have been still better.

The weighings in January and May showed that a greater improvement in weight of the underweight children occurred in the poorer sections of the city during the first four months of the current school year and that during the second four months this occurred in the better residential sections. The measles cases were fairly evenly distributed in the schools of the District, occurred earlier in the poorer than in the better sections, and therefore, would tend to bring the final weights down more in the latter sections of the city.

CONCLUSIONS

It may be concluded from these observations that:

1. There is a nutritional problem in the schools which is accentuated by the unemployment situation.
2. The children found ten per cent. or more underweight serve as a good basis for the attack of this problem.
3. About two-thirds of these children show evidences of malnutrition by physical examination.

2. Roberts, Lydia J. Nutrition Work in the Schools, 1927, page 88.

4. Observations made in the schools and homes show that there is need for stressing the necessity of providing milk for the growing child, especially when the wage-earners are unemployed.

5. That the supplying of milk in school to the undernourished children and the placing of an adequate supply of milk in the homes of families where the children were found underweight, and the wage-earner was out of work, were to a large extent responsible for the greater improvement of nutrition observed during the first four months period of the current school year.

6. That a rest period during the day, as tried out in connection with the "Noonday Health Class," is an effective means of improving the nutrition.

7. That unless seasonal variations in growth are taken into consideration in computing the expected gains in a period of less than a year, misleading results may be obtained.

8. Acute illness, which nearly always results in a loss of weight and when occurring on a large scale as during a measles epidemic, affects the nutritional status of the children during the season of its occurrence.

9. That unless all these factors can be eliminated, the simplest and most accurate way to gauge the improvement of nutrition in a given group of school children is to make the comparisons of height and weight on an annual basis.

10. Gains and loss over a shorter period than a year say, a semester, are valuable for comparing the results in such period, with the same period in another year and especially for noting the trends of individual cases.

11. In view of the fact that the opening of the schools in the fall comes at the end of the period of minimal growth, it might be less alarming to use the June weights and heights as the basis for computing the percentage of underweight children in the schools from year to year.

DISCUSSION

Dr. J. W. H. Pollard, Evanston, Ill.: I have looked forward to the privilege of listening to Dr. Koehler's paper with keen anticipation. I have been amply rewarded for my patience.

Dr. Koehler very kindly forwarded me a copy of his last annual report at the time it was issued. The completeness of his school hygiene program and the thoroughness with which all the details have been worked out immediately aroused my interest. He is to be congratulated not only on the completeness of his school hygiene set-up but also upon his foresight in developing a program to meet local conditions rather than to attempt to mold the local situation to a preconceived theoretical idea.

Dr. Koehler's deductions, resulting from the data which he has accumulated, have brought to light some very interesting facts, particularly along the lines of seasonal growth and rest in the child's physical development, the part that proper nutrition plays in this development and the unfortunate reaction, locally, of the prolonged period of economic depression.

Speaking of the depression and its reactions, as this situation developed I felt convinced that there would be a marked increase in the incidence of the acute contagious diseases resulting from lowered vitality due to malnutrition. But my surmise at that time has not been borne out, for the incidence of the contagious diseases as a whole has, I believe, been the lowest during the past year of any like period in the annals of public health.

Dr. Koehler's data on malnutrition among the Springfield school children indicates that the great majority of the children came from families who were receiving public relief. The question has arisen as to the proper balancing of the rations that the public relief officials are distributing. Since the ratio of malnutrition is not a constant, but varies from community to community, the part played by the mother in preparing the family meals might be an influencing factor. An intelligent and resourceful mother, even if the family's pantry supply is limited, will prepare a balanced diet for her children. Any mother who does not appreciate food values and what nutrition means will not blend those food-products satisfactorily from a nutritional standpoint, irrespective of any quantity and variety of foods which she may have at her disposal.

With the basic problems of a public health program, viz., sanitation, water and milk supply and the communicable diseases, now well under control in the great majority of communities, the conservation of child life comes to the front as one of the worthiest projects now being undertaken. The approach to this project varies from community to community. The sponsorship may be municipal or private. Local conditions will, naturally, largely determine the methods of procedure; but the results sought are identical. Dr. Koehler's investigations of the frequency and causes of malnutrition among the school children of Springfield and his program, already in successful operation, for their correction is an ideal illustration of one method of approach.

Conservation of child life! I don't know of any activity that is more satisfying or more apt to increasingly stimulate your enthusiasm than to feel you are doing something to protect, develop and bring up to normal manhood and womanhood the child life of our country.

Dr. Koehler, may I add my congratulations to those of your numerous friends on the excellent constructive work you are doing.

THE PRINCIPLES OF THE SURGICAL TREATMENT OF THE JAUNDICED PATIENT

JOHN A. WOLFER, M. D.

CHICAGO

There is no condition in the field of surgery which requires more skillful attention than that encountered in the jaundiced patient, if the issue is to be carried to a successful termination. So little is known of the fundamental processes involved and so much of the treatment, although beneficial, is empirical, that the entire procedure must be evolved in a methodical manner. There is no question but that an apparently hopeless case can often be restored to comparative health and comfort if sufficient skill and good judgment be exercised.

It must be accepted as a fundamental concept that those processes and conditions which produce the jaundice and are associated with it, affect not only the biliary ducts but the liver parenchyma, heart, kidneys, brain, pancreas and the ductless glands and, no doubt, all the essential organs. The mechanism of the production of jaundice is still a question. A high degree obstruction of the common duct may be accompanied with but little jaundice while profound jaundice may be associated with little or no duct obstruction; moreover, some patients who are deeply jaundiced may not be ill while others who are sub-icteric may be profoundly so. It is my belief that we are prone to estimate the condition of our jaundiced patients by the degree of pigment discoloration of the tissues rather than by a true evaluation of the condition of the essential organs and functions of the body as a whole.

Jaundice which signifies a yellow bile pigment discoloration of the body tissues, when produced by an obstructing lesion of the common or hepatic bile duct, in many instances, is associated with a group of symptoms or conditions which are of more clinical value than the discoloration of the skin. I refer to such states as the tendency to excessive bleeding, a subtle toxemia, anemia, dehydration, starvation and a mineral or chemical disbalance including the acid-base poise. These chemical changes in the body tissues are given little consideration by the average clinician but it seems reasonable to assume that they play a definite rôle in the issue.

The principles involved in the surgical management of the jaundiced patient may be grouped into those concerned with the pre-operative preparation, the operative procedure and the post-operative care. Of these I believe that the pre-operative preparation is the most important.

The subject is too large to be covered adequately in the time allocated; however, I should like to discuss in some detail a few of the more important anatomic and physiologic disturbances

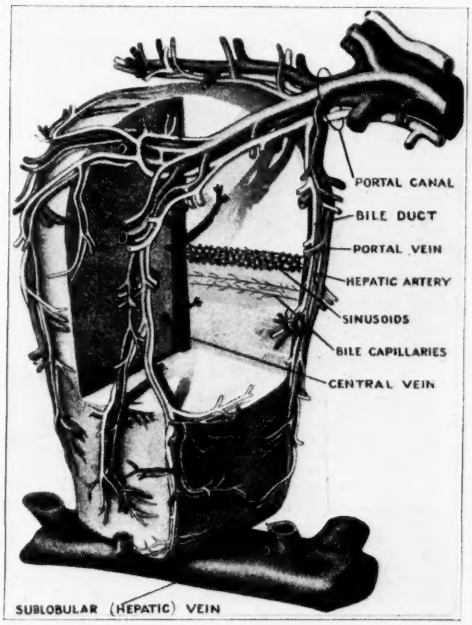


Fig. 1. Schematic Representation of a Hepatic Lobule (somewhat similar to an illustration by Braus). The relationships of the vascular and bile-duct systems and their relative size are indicated. (From Elliott and Nadler: Diseases of the Liver. Tice Practice of Medicine, Vol. 7. W. F. Pryor Company.

in the liver which may be encountered in the jaundiced patient and which, I believe, are important factors to be considered in the management of the patient.

In order to fully comprehend the rôle that the liver plays in duct obstruction it may be well to consider its microscopic anatomy. The major blood supply of the liver cells is derived from the portal blood stream. The liver lobule consists of many strands of liver cells, each strand being composed of a bile capillary surrounded by liver

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cells. In the center of the lobule is the central vein. (Fig. 1) In the periphery of the lobule there course branches of the portal vein so that with each strand of liver cells with its capillary bile duct there is formed an extensive network of blood vessels, the sinusoids. (Figures 2 and 3) The liver cells, therefore, are bathed in a liberal supply of portal blood flowing to the central vein and thence to the sublobular and into the hepatic vein. The blood flows from the periphery of the lobule toward the center while the bile in the capillaries flows from the center to periphery. The hepatic artery supplies the ducts with arterial blood. It is, therefore, apparent that any substances carried by the portal vein come into intimate contact with the liver cells. Food substances are extracted and noxious elements are detoxified. Bacteria from the intestinal tract

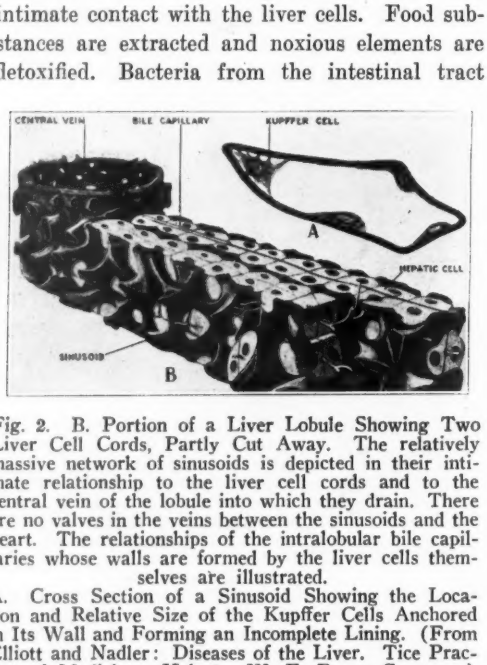

Fig. 2. B. Portion of a Liver Lobule Showing Two Liver Cell Cords, Partly Cut Away. The relatively massive network of sinusoids is depicted in their intimate relationship to the liver cell cords and to the central vein of the lobule into which they drain. There are no valves in the veins between the sinusoids and the heart. The relationships of the intralobular bile capillaries whose walls are formed by the liver cells themselves are illustrated.

A. Cross Section of a Sinusoid Showing the Location and Relative Size of the Kupffer Cells Anchored in Its Wall and Forming an Incomplete Lining. (From Elliott and Nadler: Diseases of the Liver. Tice Practice of Medicine. Vol. 7. W. F. Pryor Company.)

which may enter the portal circulation and are carried into the liver may be destroyed or filtered from the blood stream. It is little wonder that the most common organisms that are found in the liver and biliary tract including the gall-bladder are those which inhabit the intestinal tract.

Glycogen is stored in the liver cells and it is believed that an ample supply is necessary for proper function of the liver cell since Davis has shown that a liver well supplied with glycogen is refractory to large amounts of either chloroform or carbon tetrachloride. Whipple and Graham have called attention to the fact that young pups which are remarkably resistant to chloroform poisoning when starved or treated with phlorhidzin lost their resistance to chloroform. It has been shown that pups have a high liver glycogen and that starving or phlorhidzin reduces this glycogen content. Althausen believes that dextrose neutralizes many exogenous and endogenous toxins by partial or complete oxidation. Davis and others have shown that the injured liver repairs more rapidly under carbohydrate therapy. Ravdin also demonstrated this fact in experimental jaundice following duct ligation and has shown that the administration of glucose reduces the prolonged coagulation time of the blood in the jaundiced patient.

In the presence of a common or hepatic duct obstruction changes take place in the liver; some quite early, others late, the time depending upon known and unknown factors. With infection within the ducts a peripheral cholangitis with hepatitis makes its appearance; moreover, it is a known fact that when the excretory duct of a secreting organ becomes obstructed, the pressure within the duct will equal or exceed the secretory pressure of the organ. When this level is reached, the cells cease functioning and have a tendency to degenerate in spite of apparent suffcient blood supply. This phenomenon is frequently observed in the kidney with an obstructed ureter. A similar process occurs in the liver and since the cells of the liver have other functions beyond the secretion of bile it seems logical to explain phases of liver deficiency upon this basis. When no dextrose had been administered to the patient, the liver cells in the presence of obstructive jaundice as well as in cases of liver damage in cholangitis and hepatitis have been shown by Althausen to be ". . . narrow and their cytoplasm takes a dark stain. The sinusoids and bile capillaries are wide." Althausen further states: "On the other hand, the hepatic cells in section from patients who received large amounts of dextrose by mouth are literally filled with glycogen and have the appearance of those of the rabbit with 10.8 per cent. of glycogen in its liver. In these sections the large, clear intracellular spaces represent granules of glycogen and the cells themselves are so distended that the blood and bile channels are compressed." "My observations prove that even the severely injured liver in the rabbit and in man does not lose its ability to store glycogen but will do so to a marked degree when sufficient exogenous dextrose is provided. This also supports the view that the low level of hepatic glycogen in diseases of the liver is due to a shortage of carbo-

hydrate in the body caused by the slowing of the transformation of non-carbohydrate material into dextrose. When enough sugar is supplied to relieve the persistent demand on the liver for more carbohydrate, this organ at once begins to store glycogen. The fact that lipemia, ketosis and relative hypoglycemia are observed in cases of hepatic disease and also in starvation is another link in this chain of evidence that in diseases of the liver there is an internal hunger for carbohydrate."

This evidence immediately suggests a clue relative to the cause or mechanism of forms of toxemia associated with biliary duct obstruction.

The toxemia which exists in the presence of an obstructive jaundice has been studied in much detail yet the definite toxic factor has not as yet been found. It is believed by some that the bile salts are the offending substances; however, this does not account for the phenomenon present. The cause of the toxic state can be analyzed into several component factors: 1. Sepsis due to a bacterial invasion of the liver as represented by a hepatitis and cholangitis. 2. An intoxication produced by the lack of the detoxifying action of a damaged liver. 3. A deficiency state due to the lack of absorption from the intestinal tract of substances secreted by the liver in the bile under normal conditions. 4. Possibly a protein intoxication due to autolysis of the liver cells and the absorption of toxic by-products. The later is an interesting suggestion in the light of the response of the toxic patient to the ingestion of a high protein diet.

The tendency to excessive bleeding is a serious menace to many patients who are suffering from an obstructive jaundice. Little is known of the cause for the marked failure in adequate clotting of the blood; this in spite of extensive experimental investigation. The work of Ravdin throws some light on the problem so far as treatment is concerned. Hemorrhages may take place into the tissues or what is more frequent, severe and often repeated gastro-intestinal bleeding. The latter is often due to a newly formed duodenal ulcer. Various investigators have shown experimentally that when the bile is prevented from entering the duodenum, ulceration is prone to develop; this ulceration being the immediate cause of the hemorrhage. The production of the ulcer cannot be based upon the theory of alkalinization or lack of alkalinization of the duodenal contents since Blanck has shown that in the presence of a complete external biliary fistula in the dog, the introduction of sufficient bile into the stomach will prevent ulcer formation. It would appear that the cause lies in a deficiency state. The tendency to excessive bleeding is a serious handicap to any surgical procedure contemplated and unless the condition is within control, a fatal termination is to be expected. Even in those cases in which the operative procedure can be carried out without excessive bleeding, the tendency to secondary hemorrhage is exceedingly great and there is nothing more terrifying than the appearance of a general oozing from the wound or mucous membranes on the third to the fifth post-operative days. Repeated minor hemorrhages, long continued infection or toxemia, lack of proper nourishment with a vitamin deficiency, vomiting and a mineral and chemical disbalance contribute toward the production of the anemia.

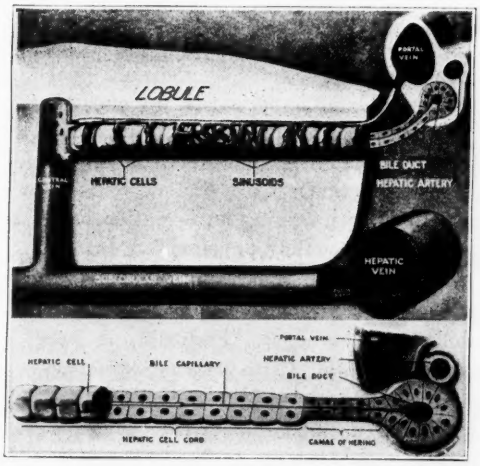

Fig. 3. (Above) Schematic Representation of a Portion of a liver lobule showing the relationships of a single liver cell cord to the bile capillary and to the venous channels.

Fig. 3. (Below) Schematic drawing of a naked liver cell cord partially cut away to show the bile capillary. Liver cells form the wall of the intralobular bile capillaries; they undergo a gradual transition to form the canal of Hering and finally the bile duct. (From Elliott and Nadler: Diseases of the Liver. Tice Practice of Medicine. Vol. 7. W. F. Pryor Company.)

Inanition and dehydration incident to excessive vomiting during acute attacks with jaundice or the inability to take sufficient nourishment or water is a preoperative condition worthy of consideration. This condition is often associated with a disturbance of the acid base balance, hence a carbondioxide combining power of the blood

plasma test should be made. It is needless to say that a state of impending alkalosis or acidosis should be corrected before an operation is attempted.

When reviewing the facts enumerated relative to the preoperative condition of the patient it becomes apparent that the principles of the surgical management of the jaundiced patient resolve themselves into an adequate preoperative regime, the removal of the obstruction and an adequate post-operative control.

It has been shown that the liver demands sugar or rather gylcogen for proper function, also that in the event of starvation or liver damage, the liver glycogen is reduced. Further it has been established that the damaged liver can convert exogenously given dextrose into glycogen and store it in the liver; therefore, sugar should be administered in amounts sufficient to restore liver glycogen. The administration should continue over a period of days, the time being decided upon evidence presented by the individual case. The more toxic the patient is, the more time, as a rule, must be consumed in restoring liver glycogen. I should like to reiterate my frequently expressed dictum: "Do not allow yourself to be rushed into performing an operation before the patient is adequately prepared."

The method of sugar administration will depend upon the condition of the patient and his ability to take sugar. Preferably it should be given by mouth. It has been my experience that sugar introduced into the gastro-intestinal tract will build up an adequate liver glycogen more rapidly and more surely than when it is given in any other way. Sugar may be given in the form of a high carbohydrate diet giving such foods as rice, potatoes, sweet fruits aided by candy, karo syrup, honey and cane sugar. The amounts depend somewhat upon the individual—150 to 300 grams daily. In some instances even more will be tolerated by the patient. It is well to remember that the amount should not be pushed to the extent that sugars become nauseating to the patient, the tolerance should be built up gradually. When vomiting is present or if the patient cannot take the required amount by mouth, dextrose or glucose should be given intravenously. When given intravenously, the sugar is introduced directly into the blood stream and at times a considerable amount is wasted since portions may be eliminated by the kidneys. When given into the gastro-intestinal tract, the absorption is slower and time is allowed for conversion into glycogen; however, there are times when the glycogenetic process is embarrassed and a glycosuria is present. A word of warning may be issued relative to the use of insulin under such conditions. It seems rational to suppose that in the presence of a glycosuria in a devitalized patient with liver damage insulin might be indicated. These patients do not respond well to insulin. I have had two patients recently in which a profound hypoglycemic shock was precipitated with 5 units of insulin. Recent work on the action of the hypophysis and the hypothalamus in carbohydrate metabolism may eventually throw some light upon the subject. Glucose may be given intravenously in solutions from 5 to 50 per cent. depending upon the necessity for water. We rarely use the more concentrated solutions, depending upon 10 per cent. dilution.

There is no test that I am familiar with which will indicate a re-establishment of the liver glycogen. Theoretically the bromsulphthalein test should give us definite evidence; however, it is known that even in the presence of extensive liver damage, the remaining portion of liver tissue may eliminate the dye. This is another evidence of the "safety factor" of essential organs. It is my conviction that in the event of a markedly increased bleeding and coagulation time, the appearance of a bleeding and coagulation time within the normal following the repeated administrations of glucose is the best criterion available at the present time. In the presence of a complete obstruction of the duct and in the event that glucose administrations and blood transfusions do not accomplish the desired purpose, a rapid decompression operation—drainage of the gallbladder or duct—should be done and the administration of sugar continued without an interval. This procedure should always be preceded by a blood transfusion. When the condition of the patient has improved sufficiently, the more extensive operation may then be undertaken with considerable hope for success.

During the preparation of the patient the protein diet should be basal since it has been shown that the obstructed patient tolerates proteins badly, in fact, a fatal result can be precipitated by a relatively high protein diet.

In the event of an anemia or when the red blood cell count is below 3,000,000 one or more blood transfusions should be given during the time the patient is receiving glucose. Sufficient water must be given at all times and when the patient cannot take it by mouth, subcutaneous administration of physiologic salt solution or Ringer's solution must be resorted to. Care must be exercised that too much salt is not given the patient with damaged kidneys, leading to a salt retention. This can be prevented by the use of 5 per cent. glucose solution instead of salt solution.

If the patient has continual vomiting over a period of time so that the element of starvation is in evidence, the problem at once becomes more complex. Under such conditions the toxemia may continue even though the bleeding time may be within the normal range. When such a circumstance exists I believe that jejunal feeding is of decided benefit. Seldom will a duodenal tube pass through the pylorus because of spasm. A rapid laparotomy with implantation of a tube into the jejunum can be done in most instances and at once fluids may be introduced into the gut through the tube. Within 12 hours a specially prepared pabulum can be given and within a few days the patient can be fed daily 3000 calories in the form of a properly balanced diet. In this manner water, nutritional, mineral and vitamin balance may be established and maintained over a period of time with no inconvenience to the patient. I believe that this procedure is a life saving measure in selected cases.

So far as the operative procedure is concerned I have little to say except to urge the use of two stage operation more frequently. I believe that the error often committed in common duct surgery is that too much operative work is done in an effort to correct the parts involved. These patients do not tolerate long operative procedures unless unusually well prepared. Any abuse or exposure of the liver may precipitate a physiologic breakdown; moreover a secondary kidney failure due to either the prolonged or severe toxemia may intervene. In the event stones are removed from the common duct, I believe it wise to drain the duct so as to prevent a secondary obstruction by edema. In spite of the short duration of this secondary obstruction it may be the lethal factor in a patient who is still critically ill and who has just been subjected to a devitalizing operative procedure.

Time does not permit a discussion of the details of the operative procedures. It must suffice to say that the keenest surgical judgment must be exercised at all times.

The post-operative care of the jaundiced patient is no less important than the pre-operative. It is my honest conviction that patients may be efficiently treated from an operative standpoint and yet a fatal termination ensue because of a lack of an understanding of the post-operative management. Liver glycogen must be maintained by the continued use of suger. In the anemic patient and especially if considerable blood has been lost during the operation a transfusion should be given immediately after the operation. The patient must not become dehydrated. If nausea and vomiting persist, gastric lavage should be done and if a considerable amount of gastric contents is evacuated, the tube should be left in position and the stomach kept empty by frequent aspirations. Great care must be exercised during the first post-operative week since the patient is combating not only the disease but the results of the operation. The state of nutrition, water balance, sugar intake and the loss of vital substances such as minerals and chlorine must be constantly observed.

In the presence of a total external biliary fistula it may become necessary to introduce bile into the gastro-intestinal tract. This may be accomplished with the stomach tube or bile may be given in grape juice by mouth. In the presence of a jejunostomy, emulsified bile may be introduced through the jejunal tube.

A word relative to "High Temperature Deaths" following gallbladder and biliary duct surgery may be in order. This bizarre post-operative tragedy, in many instances, gives little warning and may occur in a patient who appeared to be in good pre-operative condition. The state preceding death has the characteristics of an allergic reaction so profound as to destroy life before it can be combated by an internal mechanism. I have seen far less of this condition since I have prepared the patient more efficiently pre-operatively.

In conclusion I should like to emphasize several points in the management of the jaundiced patient: 1. Adequate pre-operative preparation

of every patient along the line presented in this paper. 2. Judicial and meticulous surgical technique and judgment. 3. Efficient post-operative management which is identical with the pre-operative preparation. Close adherence to these principles will markedly reduce the mortality in biliary surgery.

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DISCUSSION

Dr. H. A. Bollaert, East Moline: I should like to ask Dr. Wolfer to what he attributes these allergic reactions which he mentioned. I wonder how he explains them.

Dr. John A. Wolfer, Chicago (closing): Relative to Dr. Bollaert's question, there are many possibilities. No doubt the operative trauma to a damaged liver is a factor. If we look at it in this way: we have a badly damaged liver whether due to disease or secondary effects of obstruction; we will assume that the glycogen content is very low and we add to that the trauma of the operation. We wonder about some of Crile's theories, but they are not theories when he speaks about lowering the temperature of the liver with operation. Some years ago he used diathermy to keep up the temperature of the liver. It is possible that the exposure of the badly damaged liver to a lower temperature, together with the foreign proteins which are liberated during the operation, tend to bring on this allergic reaction. There is something that is set off like dynamite and an explosion occurs. We have used adrenalin and once in a while brought the blood pressure back, but most of the time it has had no effect and the patient died. I do not think the cause of death in these cases has ever been proved but some time perhaps we may find it.

RHEUMATIC HEART DISEASE IN SCHOOL CHILDREN

RAY E. LOGAN, M. D.

GALENA, ILL.

The basis of this discussion of rheumatic heart disease in school children rests upon an actual clinical experience covering six years of observation of cardiacs in the elementary schools of the city of Detroit. Our statistics have been obtained practically wholly from an average number of 471 children, who were followed in their work during the school years of 1928 to 1931, inclusive. Only a small group was seen in the high schools and none in the parochials. This work was done by myself, with the assistance of one full time nurse assigned to the work and with the assistance that regular school nurses could give us. It was impossible, therefor, to make use of all records as a number of these were incomplete in some detail. Our conclusions are made from groups of fully completed histories and records, making a composite picture. Our findings are corroborative of other observers of large groups of children with heart disease. We have taken freely of extracts from the literature in comparison with our study.

TABLE 1

Totals of number of children examined, admitted and studied Detroit Elementary Schools:

	1928	1929	1930	1931
Children referred for exam.....	603	721	4,364	800
Class I-IIa-IIb-E-F — Children admitted	420	403	635	525
Number studied	420	142	635	525
Number of organic Rh. heart.....	305	101	378	335
Per cent of Rh. heart to cases admitted	72.6	...	61.1	63.8

The average number of children in all the elementary schools of Detroit during the years 1928 to 1931, inclusive, was 236,645. Our work included only children referred from this age group. Presumably a relatively large number were not referred, for various reasons. An average of 339 rheumatic valvular heart disease cases were in school each year. This would give an estimated rate of 1.4 organic rheumatic hearts per 1000. This definitely low rate is probably due to the reasons already given, and because that while the parochial schools were included in the total enumeration, yet we have no accurate data as to the actual number of cardiacs in the

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latter schools. In examining 2317 children in a regular school in an industrial center, we found 8 cases of organic heart disease, a rate of about 4 per 1000, none of whom were known previously. Nine cases of organic heart disease were found in a suspicious group of 117 in four high schools. Only one of these had been under observation in the elementary grades. From this data we believe that, 5 to 6 per 1000 can be safely assumed to be the rate of organic heart defects in Detroit schools. This compares favorably with the figures of 6 to 7 per 1000, found in examination of the entire school population in Boston and New York City, and of 7 per 1000 in 598,167 children in Wales (1924). It is likely true that no school survey can be 100 per cent accurate, owing to absentees, children ill at home and in hospitals. Furthermore, it is evident from the large number of functional hearts erroneously referred as organic cases, that the examination of such children must be done with regard to a uniform method of procedure to obtain acceptable diagnoses.

The material for our study of the rheumatic heart problem was obtained from referrals of true and suspected rheumatic infection and rheumatic heart disease, by private physicians, clinics, welfare organizations, and from the nursing inspection service of the department of health. Thus we had children from all over the city placed in more than 60 units where special rooms were set aside, as well as a number of small schools built with especial regard to ventilation and sunlight for the care of undernourished, convalescents, tuberculosis contacts and deformed children. The large number of rheumatic histories; children with possible and organic heart disease found in those admitted for this building up program, prompted the forming of cardiac groups as well. This was the beginning of the school care of heart cases. On the average, during my work, about 100 of the heart group were transported each year by a bus to and from school. The object of the care of rheumatics was to furnish a longer period of convalescence, lessen the school schedule burden, give the benefit of fresh air and periods of rest and permit a better supervision of activities at home and school.

That rheumatic infection and its accompanying cardiac involvement is largely an urban problem is quite definitely established. True, there is no literature on the prevalence in smaller towns or in rural districts, but undoubtedly, accurately kept records of physicians in these localities would show a surprising number. Without question the factors of overcrowding, vitiated air, poor housing, unbalanced diets, climate, etc., are predispositions to the disease. Wherever the disease is found, it is certain that rheumatic infection is fundamentally a problem of childhood, and a study of heart disease in children resolves itself into a study of that infection. In onset at an early age, progressiveness of anatomical changes, recrudescences and chronicity it is strikingly similar to tuberculosis and attention to this was called as early as 1888, by Garrod.

The recognition in the past few years of the fact that rheumatic fever may manifest itself in a variety of forms, with general systemic manifestations, rather than local; with insidious onset rather than fulminant, makes it desirable to recognize this as a definite disease entity and has rendered the term rheumatic infection more fitting than the former. It should be remembered that in children arthritic symptoms are comparatively uncommon. We found that the finding of a heart defect was the first indication of rheumatic infection in a large percentage of the children.

The etiological organism of the disease is undetermined. Suspicion rests quite solidly on the streptococci. Apparently certain children are more prone to infection than others. This may be due to natural immunity as in other acute infections, or as Swift's work would suggest, the possibility of a specific sensitivity to certain of the streptococci. The influence of hereditary predisposition is quite probable as in tuberculosis and much evidence may be found to indicate infection spread through contact. Histories of 525 children in the schools in 1931 show that 100 children had family contact with rheumatic infection. Two children in one family were in school with rheumatic heart disease and four in one family at our clinic at the Grace hospital. Review of the literature will indicate that these figures are not mere accidents.

The influence of sex, race and focal infection are of interest. A review of our records for

the three years of 1928-30-31 showed that 56 per cent. of the total case load were girls and 44 per cent. boys; 30 per cent. of these came from homes rated as economically good to excellent and 70 per cent. from mediocre to poor. The children of Polish, Jewish and Italian parentage predominated in the order named. Contagious diseases and foci of infection are known to be the apparent inciting cause of a rheumatic attack and the general feeling is that defective teeth, tonsils and infected sinuses should be eradicated. Of 525 children in 1931 there was a history of tonsillitis in 228; 273 had been tonsillectomized; 67 had rheumatic infection after tonsillectomy. The histories suggested that the first recognized attack followed scarlet fever in 28 and tonsillitis in 138. One case definitely was due to an infected tooth. Presumably the school health program with attention to prevention of contagious disease and removal of infectious foci has had an influence, for Glover in England and Atwater in the United States have shown that there is a marked fall in rheumatic infection in the past 25 years, and the *Illinois Health Messenger* of Feb. 1, 1934, states that the death rate from heart disease in the ages below 20 was less than half that for the same age group, in 1922.

The lack of epidemiology and the too often inaccurately reported deaths from heart disease, makes it difficult to determine the incidence of rheumatic infection. It has been estimated to make up 3 to 7 per cent. of the admissions to general hospitals both in the United States and Europe, in certain regions. It is a disease of high morbidity due to its predilection to heart involvement especially in young children, but without this complication it has a low mortality (about 1%). In children, even the mildest attack may cause a cardiac involvement. This anatomical change may be the first evidence of the existence of acute rheumatism. Dependent upon the valve affected, the extent of the lesion, its activity and the convalescent care, the heart suffers permanent damage with a loss of efficiency and a certain amount of accompanying disability. Acquired heart disease is, in conservative figures, 65 to 80 per cent. of rheumatic origin in all ages and in children is probably nearer to 90 per cent. The mortality rate in children seems to be found at 10 to 12 per cent. and, as Stroud remarks, is about the same over a 10 year period, in spite of the care given. In a three year period the death rate in Detroit schools was 3.4 per cent.

It is but a comparatively short time since it was considered useless to attempt to do anything definite for children with heart disease. However, results of prolonged convalescent care with supervision of the physical activities, removal of foci of infection and building up the natural resistance of the child has yielded promising results. The education of the child makes it necessary for the school to assume some of this problem. School programs for the cardiacs are being carried on in New York, Chicago, Boston, Philadelphia and Detroit. Convalescent care with school work is being done, notably in St. Louis, Philadelphia, New York and in England. The results have been very gratifying.

If an attempt is made to supervise cardiacs in the schools it is well to know the type and extent of the anatomical changes. To make a diagnosis of organic heart disease we adopted the same criteria for diagnosis as used in the Boston survey, i. e.—

Any definite murmur not a third sound, diastolic in time.

A clear enlargement of the heart.

A systolic murmur, definite and persistent.

Any one of the above was accepted as indicative of organic heart change. For the etiological diagnosis, rheumatic infection was accepted from history of arthritis, repeated tonsillitis, chorea, growing pains of duration, and unexplained fever, fatigue and subnormal weight,—and the type of valve defect.

Analysis of 328 definite organic hearts observed over a period of four years showed the mitral lesions were 70.5%, aortic 6.6%, mitral and aortic 6.9%. As a matter of comparison and interest, congenitals were 16%. Approximately 10% showed extreme cardiac enlargement. The type of lesion has a bearing on prognosis. Mitral insufficiency, which is the most commonly found, gives the least discomfort if there is no active infection. Mitral stenosis complicates the picture and these children are more prone to discomfort and to respiratory infection. The degree of hypertrophy, as might be expected, is an index of value in determining the expectancy of myocardial insufficiency. The prognosis of these heart conditions depends of course largely on the state of the myocardium. There were very few

abnormal rhythms associated with this organic heart group. Extrasystoles were quite frequent although transient, while auricular fibrillation occurred in only a few before death, as noted by Stroud and others

The age of the initial attack of rheumatic infection or at least the recognition of the heart

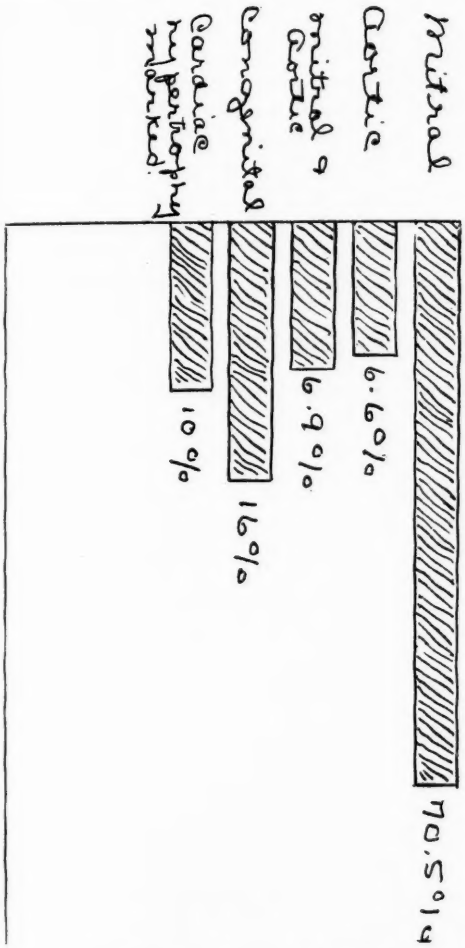

Chart 1. Percentage of anatomical cardiac valvular lesions found in average number of 328 school children (Detroit) during 4 years' special observation.

involvement is of value, owing to the more accurate determination of probable recurrences of infection. 211 histories were studied for the onset age. 99% occurred before the age of 15 —70% being between the ages of five and ten. The average age was 7.4 years. Wilson, Ling and Croxford found 98% before the age of 15, the average age being 7.3 years. Mackie reporting 366 cases among children found 78% between five and ten years.

The difficulty of making an estimate of a child's chances in school or of his having a recurrence of his original infection, is based on the well recognized characteristic of rheumatic infection to suddenly become reactivated, both systematically and in the endocardium. This one feature alone makes prognosis necessarily guarded. 75 children in the group of 211 gave a history of 1 to 5 recurrences, amounting to 100 attacks. 49% of these occurred in the first two years after onset. 34% came in the 3rd, 4th, and 5th years, with none after the 9th year. We found the greatest number of these recurrences between the ages of 8 and 12 years. A decrease in the probability of reinfection has been found

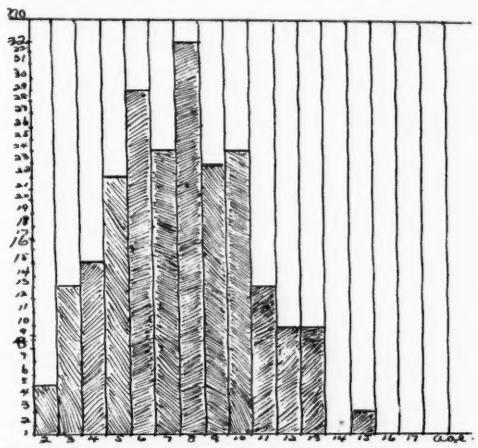

Chart 2. Incidence—Ages of onset. 211 children. Average age 7.4 years.

by all observers, after the 14th year. Mackie reporting 252 cases found 58.8% before the 4th year after onset with the greatest number, between five and ten years of age. He believes that recurrences will occur in 71% of rheumatic infection and may occur at any time regardless of age with the greatest probability of doing so, 1 to 4 years after the initial infection.

It will be found necessary to exclude certain children with rheumatic heart disease from school, because of the probability of danger of infection to others, to furnish the necessary rest for the heart or to build up resistance against an intercurrent infection. Most of the exclusions occur in the early winter months and in March and April. Undoubtedly this is due to the prevalence at these times of acute upper

respiratory infections. A number of our exclusions were found necessary because of low grade fever, fatigue and loss of weight in a child whose functional cardiac status may have been good.

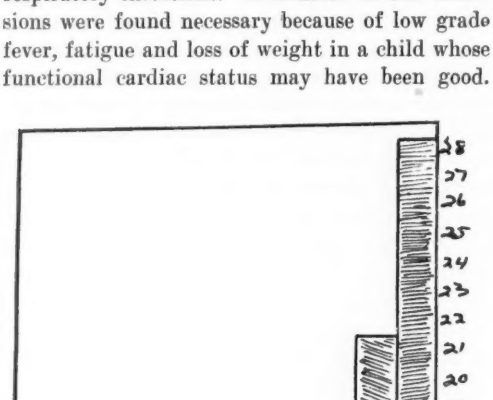

Chart 3. Incidence of recurrence in years after initial attack. 100 recurrences in 75 of 211 children.

These findings undoubtedly indicate latent rheumatic infection and are the best criteria to adopt in school. In spite of the fact that in Detroit the cardiacs are given an easy schedule of work, that rest periods in school hours were enforced and intermediate feedings given, at least 8 to 10% were excluded each year for serious trouble, while 12 to 15% more were out for shorter periods of time; 10% were unable to finish school during the year of 1930, the largest number of any of the four years. Eight of these died. The fear of death at school is one of the annoyances of school work. Teachers and pupils alike voice this dread and makes it more difficult for the cardiac victim to escape a marked psychic unbalance. As a matter of fact, there is but little danger of such a happening. I do not find any record of a death in school in the entire four year period. It may be said that in children, infection is the main reason for cardiac breakdown, and that exertion within reason is a minor factor.

In order to systematically regulate the activities of any group of cardiacs, it is necessary to rate each one according to some standard. We have such a standard in the Cardiac Clinical Classification, as recommended by the American Heart Association. Briefly this is:

Class 1. Organic heart cases able to carry on ordinary physical activities without discomfort.
Class 2. Organic heart cases unable to carry on ordinary physical activities without discomfort.
A. Slightly decreased.
B. Greatly decreased. Evidence of activity, etc.
Class 3. Unable to carry on at all.
Class E. Possible heart lesions for observation.
Class F. Potential infections that may lead to organic change.

During the four school years those with anatomical defects were classified as 49.5% Class 1; 32.9% Class 2A and 4.7% Class 2B. Of those without definite anatomical change 13.6% were Class F. Possible heart disease or Class E are not considered here. Since Class 1 and 2A are apparently well children, then more than 80% of our children were fit for school. But reinfection may occur and classification of function is changed thereby. Class F, unless free of symptoms, were not entered in the school. It was the policy in Detroit to admit a few of Class 2B, but this is not a wise plan as pointed

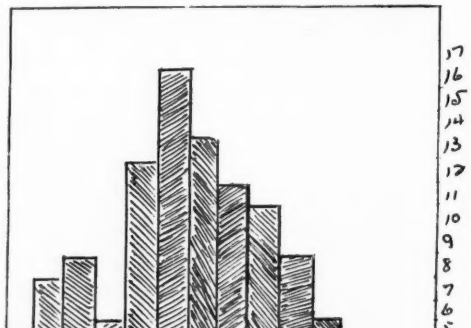

Chart 4. Table showing incidence of ages of recurrences. 75 children, 100 recurrences.

out by Halsey, who believes that exclusion of all 2B children is a therapeutic measure of prevention and a measure of conservatism for their benefit, as well as a measure of prevention of infection of the well children. Class 3 in school is out of the question. It is possible to furnish home teaching to certain of this group.

It might be expected that children afflicted with heart disease would be irregular in school attendance and such is the case. As is well said by Foster, in his discussion of the psychic factors of heart disease, "Whether children with heart disease fall behind their age in school through interruptions due to illness or to lowering of energy, it has been found that these children do show a certain diffidence and lack of initiative in school." Children with a heart affection naturally must miss a year or so of school work. Accordingly, when they are able to return, they feel the loss of this time more keenly than we may know. Also the nature of the affliction makes them the favored one at home and the favored and feared among the teachers. He also must endure the remarks of his thoughtless little friends about his inability or unwillingness to join them in the usual activities. Thus the child develops a warped personality which while not true alone for heart disease, is however, well marked in this group. The psychic unbalance of children with heart disease should be well understood by teachers as it is a field well worth while.

Whatever the cause, children with heart disease find it comparatively easy to find reasons for absences. In a study of the reasons for absence in New York City among a large group, 63% of these were found to be in no way connected with defects of the heart. In Detroit in one school room we kept the records of 42 children for one semester. We found 6 absences because of heart trouble; 19 were for acute colds and minor illness and 88 for reasons in no way necessary because of heart or health.

Since an education is vital for the cardiac child in order to give him the best chance at life without hard labor, this loss of interest in school is tragic. We attempted to overcome this by explaining to the children old enough to understand, their need for education in view of the disease, and that they could do well in spite of the affliction if they chose to do so. Natural inclinations were favored toward some special objective. In spite of interest of teachers and nurses alike, a large number of our children dropped out of school after finishing the elementary grades.

That children with organic heart disease can make a perfect score in attendance is possible. We followed 38 children with organic hearts for one year in regular grade rooms who were allowed to do everything except competitive exercise and gym. No illness, no deaths occurred and no exclusions were necessary. These were all Class 1 and Class 2A. In comparison with the same class under careful supervision it would appear that, granted the cardiac lesion is quiescent, that regular school routine is as safe for them, as is special care.

With the exception of a majority of the congenital cases, children with organic heart disease are as bright mentally as the average child. I think it is safe to say that the instruction of the child with heart disease offers no more difficulties than that of the normal child.

Our observation of children with rheumatic heart disease in school and the studies of other large groups of children in the United States is indicative that the problem of the cardiac in school is one of considerable proportions. The prevalence of rheumatic infection and of heart disease, in the younger age group, the extremely probable infectious nature of the disease, make some recognition of the problem necessary, not only for the victim but for the good of the well children. The resemblance to tuberculosis in children is apparent, and the same routine now observed in tuberculosis may well be fitted to rheumatic heart disease.

Experience would indicate that children with rheumatic heart infection should not be admitted to school until cardiac function is definitely Class 1 or 2A, and even then the possibility of recurrence of infection during the first four years must be borne in mind. If such classes only are admitted, it would appear that they may well attend regular schools, with some restriction and observation by the school nurse and by their family physician.

Prolonged convalescent care in sanatoria has given excellent results and suggests the need for more extensive public health interest along these lines. To quote Schlesinger, "The pre-

vention of rheumatic heart disease in children is a national problem and will only be solved eventually by national resources."

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DISCUSSION

Dr. Gerald Cline, Bloomington, Ill.: We have listened to a most wonderful paper on rheumatic heart disease in children. If I were to classify a rheumatic case, I would call him the neglected or forgotten child. So often we do not see them until they are bed-ridden. The ordinary clicking or murmuring heart of the child is not picked up until he has a definite picture of a cardiac embarrassment.

We do not see as many children with rheumatic heart disease in a smaller community like Bloomington as the Doctor has quoted from the Detroit School mainly because our children do not have as careful physical examinations.

I have a few questions that I wish to ask the Doctor: 1. I wonder if he has the experience that the allergic, rheumatic child is a little more prone to be a cardiac case than a non-allergic child. 2. What has been his experience with the sedimentation test in regard to an index for allowing the child out of bed and returning to school. Drs. Black and Elghammer in Chicago have done quite a bit of work with this sedimentation test and enthusiastically report its reliability.

Dr. Logan's paper has brought out the important point of an early diagnosis.

The three most important symptoms or complaints, namely fatigue, leg aches and night cries, must again be kept in mind. Night cries might be dreams but always must be kept in mind as the first signs of impending rheumatism.

The rheumatic child with pancarditis shows quite marked cardiac symptoms without fever yet so often we have been taught to watch the fever in these cases. Another point is that the child so often has a tender, slightly enlarged liver, much more commonly than they have the early edema which is so common in adult cases. Pallor, cyanosis and albuminuria all seem to precede edema in children.

Dr. Logan has shown us that scarlet fever is so often a forerunner of rheumatism in children particularly in cardiac cases. However, I think we sometimes lose track of the ordinary little common cold, whooping cough and particularly measles which have been so frequent throughout the State this year, as much more causative agents of rheumatic heart disease. No matter how mild these diseases are, often they leave a path of complications that is so worthy of consideration.

Pay attention to the teeth first because the ordinary little gum boil around a deciduous tooth is often left uncared for with the idea of allowing the second teeth to erupt in better alignment. This finding seems to me perhaps more commonly the cause of rheumatism than tonsils.

Dr. Gottfried Koehler, Springfield, Ill.: In connection with my work here for two years with the Springfield schools, I am convinced at the present time that the care of the children in the schools with heart disease, in at least this community, is more important than the care of the crippled children. We try to examine the children in the first, fourth and seventh grades in the elementary schools. I dare say that our heart cases run pretty close to one per cent.

I took this matter, of the special care of children afflicted with heart disease, up with the Attorney General of the State last fall. There is a provision in this State for taking care of the crippled children and for taking care of the children with markedly impaired vision. In these cases the State pays the difference in the cost of educating these children in the special school as compared with educating them in the general classes. We had gradually been sending our heart cases to the crippled children's school and the sunshine school. The Attorney General went into the matter very thoroughly and one of his assistants prepared a preliminary opinion to the effect that a heart case was not a crippled child. Mr. Kerner, the Attorney General, was very kind about the matter. He showed me the original opinion, and we had an opportunity to present a brief, with a view to showing that a heart disease case was a crippled case. But he finally ruled that they could not legally be classed as crippled cases. We took this matter up through the Superintendent of Education. I have always regretted that we didn't take it up through the State Health Department.

When we go into the etiology of the cases, we find rheumatic infections manifesting themselves, as Doctor Logan has said, in a majority of the cases. We frequently find this syndrome of tonsil trouble, not necessarily large tonsils, manifesting itself in repeated attacks of tonsillitis with so-called growing pains. In quite a percentage of cases I have found that pneumonia has apparently been a cause; pneumonia in infancy, complicating measles or some of the acute infections, diseases, or just ordinary pneumonia.

We find cases of heart disease in our high school gymnasium. We require a physical examination before there is any participation in athletics. We get a great

many requests from parents to excuse their children from "gym." I dare say that in very few of these do we find heart disease. In our program for next year, we plan to make sure that the child participating in physical education, even in the elementary schools, is examined for heart conditions.

I think in the smaller communities we should work towards classifying these children as crippled children, so that they can be taken care of in the same way that the crippled children are taken care of.

On account of the fact that we haven't had an epidemic of infantile paralysis in this State recently, our crippled children are becoming fewer.

I don't believe it is a good idea to put these children in the same room with crippled children but, as a matter of necessity, it must be done in certain cases, and it probably is the economical way of taking care of these cases in the small communities. That gives these children the transportation that they need, it gives them the rest periods and everything else that is necessary to build up their strength. We have had cases in the schools that couldn't climb the stairs. I remember one child that was being carried upstairs. Yet, these children attempt to attend school.

Therefore, I say that this paper is a timely one and I hope that in Illinois the present law relating to the education of crippled children will be amended so that it will cover heart disease cases.

Dr. John P. Coughlin, Chicago: Dr. Cline said that the cardiac case, the rheumatic cardiac case was the forgotten child.

I would like to make a plea here for another type of child that we don't see very much of, but they die around sixteen or seventeen years of age. That is the older child or the young adult who has left school—and there are a great many of them now—because of economic conditions.

I have noticed that those children who are free from reinfections over a period of four to six years can then have infections and it apparently doesn't seem to disturb them, but if they have a second infection at the end of six months or a year, or two years, invariably they have a lighting up of their endocarditis.

The problem of psychic unbalance that he mentioned is very interesting. I don't believe they have connected up the factor of rheumatic endocarditis with this psychic unbalance. I believe that we should take these young people—girls, especially—when the are twelve or thirteen years of age and sit down and talk to them. If we tell them that they are not going to die, that they will live to be old women, that they will ge along as well as anybody else, and explain to them what has happened to their hearts, what can happen for good and what can happen for evil, I think we can straighten out that psychic disturbance. I believe we ought to remove any and every focus of infection that we think can have anything to do with the heart.

Dr. Ray E. Logan, Galena, Ill.: I am grateful to the doctors for their kind expressions. I am very glad you have enjoyed the paper. This paper was written for the very definite purpose of bringing out discussion. It was written with the hope that some thing would be done for, as the doctors have said, these neglected children; because if there is any class of children who are neglected, it is the cardiacs. I feel this probably more deeply than any one else in cardiac work because I lost my own wife some four or five years ago with a heart lesion which, if she had the proper care when she was fifteen years old, she might have managed to escape the death that did occur. So, you see, I feel very deeply.

Then, I feel very deeply also for the work I do with these children because, if there is anything that gets into your heart, it is working with a group of children in school, and see them leave, and some of them get better. So, I think this work in the schools is very worth while.

As far as the allergic reaction is concerned, doctors, we didn't do any research work on this whatever. We started out to do a five-year study of heart disease but, unfortunately, old man Depression came along and raised the devil with our program, and that is why I am back in the State of Illinois. We did have some definite allergic reaction in the abdomen, but I didn't say anything about some of these symptoms because this subject is so vast that it is practically impossible to cover it all. We had some very definite reactions in the cardiac clinic of Grace hospital, which I was fortunate enough to be associated with. We had a good many school children turned over to our cardiac clinic, and there we had about two very definite allergic reactions in the abdomen which simulated an attack of appendcitis. We didn't, however, do any allergic tests; we didn't do any sedimentation test. We were prepared to do a whole lot of tests but, as I said, the depression came along. We had several cases of adhesive pericarditis in which the chest wall bulged out almost as large as my fist lying upon the chest. In four of these we were lucky enough to be able to get the consent of the parents and did a decompression operation, and three of those children were living a year later. They were very much improved. We didn't rely on any symptoms such as edema of the ankles in our school work. We relied upon loss of weight, fever, lack of energy and probably some attacks of arthritis, although arthritis was a very uncommon finding.

I expected when I said that mitral insufficiency was one of the most common lesions in children that you would say something against that because I do feel that, while a large number of these children show what we call mitral insufficiency, I have a grave doubt that there is such a thing as a definite simple mitral insufficiency. In other words, I believe that mitral stenosis is practically always associated with it.

In the State of Michigan, cardiacs are recognized as cripples and as such given vocational training and we were lucky enough to get some of our children put in vocational training. They were recognized as cardiacs in so much as they were not given any work in the factories there on account of the industrial hazard.

Finally, I want to say one more word about the psychic unbalance. I was glad to have the doctor say something about that, and about the fact that the children at the age of twelve or thirteen have this phychic unbalance. That is a subject all in itself, but there are

several features of this paper that you could talk about and use as a discussion in themselves. That was just a part of the paper. You could take a whole hour on that one thing alone. Psychic unbalance is one of the most unfortunate things we have to contend with. I might tell you many, many things about how children were practically carried upstairs and were not allowed to do this or that; they were made nervous wrecks practically, especially those towards the age of adolescence. And there is only one way to get out of it, gentlemen. When you come across these heart cases, take your youngsters when they get around the age of fourteen, fifteen or sixteen, and sit down and have a good talk with them, and then take the father and mother off in a corner and have a good talk with them too, separate from the child, because as usual, ninety per cent of all the troubles mentally of the child the parents are responsible for.

I certainly want to thank the doctors very much for their interest in the paper.

Dr. Gottfried Koehler, Springfield, Ill.: I recall reading a couple of months ago, I think in the Journal A. M. A., an article on what became of these cardiac cases in the schools. My recollection was that the answer to that question was that over 30 per cent. of them died before they reached the high school. I was rather surprised at that. Is it your information that a large percentage of them die at that early age?

Dr. Ray E. Logan, Galena, Ill.: No. As a matter of fact, we don't know that. Our death rate in the ages between 5 and 16 was only 3.4 per cent. in the three years. If you will look up Stroud's paper in The Journal A. M. A. sometime last year you will find some good statistics on the death rate of heart disease in children. Over a ten-year period it ran about 10 to 12 per cent. As I told you, he said in spite of the care given, it would do that. Here is an interesting fact; initial infection occurs in 78 per cent. of cases between the ages of five and ten years of age. If it strikes at five, you are practically certain, if the child doesn't have a recurrence before the end of four years, and he does have another recurrence after that, it will not injure the heart as if it had come in six months or a year. You will find the literature will tell you the same thing all the way through.

We had two girls, very bright; one was a Negro girl and one a Polish girl. They graduated from the elementary school at the age of sixteen with honors. They went to high school. In their first year in high school both of them, having been kept down very carefully during the elementary grades, where they hadn't the usual love affairs that the average girl has during her regular school period in the regular grades, both of them fell victims to a love affair. Both were married; one was married openly and the other was married secretly. Both of them became pregnant and both those girls died. One died in six months and the other in eight months, which shows that pregnancy at that age with heart disease is much more serious than in the same age without.

So I say that, if the child gets beyond his four year period in school without any reaction, and you can carry him along during that time, I think his chances of getting along after that are very good.

If I had the power of saying in the State of Illinois when a child should go to school, I would say that the child who had a definite rheumatic infection, who had developed a heart lesion at the age of between five and ten, should not be allowed in school before three or four years after his infection had occurred; and he should be kept under very careful systematically graded exercises, and I believe in that way we can prevent it.

And that brings us to sanitarium care and prolonged convalescent care, and that is the object of this paper largely, in the hope that some seed would be sown today that would lead eventually in this big State, when times get good again, that we can have some sanitarium or something in which we can put these neglected children who are certainly as much crippled, or more so, than if they had a leg or an arm off. If I lost a leg, the State might take care of me for life. But if you lose a heart, the State will say, "It's too bad. We can't do anything for you," and they let you go on about your business and allow you to pass on.

Dr. G. W. Haan, Aurora, Ill.: You mentioned this matter of the psychic element. Where do the parents find out that there is any disease? From the doctors, don't they?

Dr. Logan: Yes.

Dr. Haan: The doctor says there is a heart leak, there's an enlarged heart; there's a valve injured. Why should the doctor tell such things as long as the heart is functioning well, as long as it responds well to effort?

MacKenzie, who probably knew as much about heart diseases as most of us do, made the blunt statement that nobody ever died from mitral regurgitation. Then we tell the people that there is a heart leak. The doctors should be more considerate what they say about it and then the psychic element need not to be considered.

Dr. Herrick made the statement that we were calling a great many heart diseases dangerous when they are not dangerous at all. I happen to receive a good many reports of deaths in my capacity as health commissioner, and sometimes more or less I feel that there is not enough study made in writing out a death certificate.

Myocarditis is so easy to say. There is no appeal from it. It settles the question. He is the last fellow that sees the patient. And, myocarditis doesn't need any explanation. I think sometimes it needs a great deal of explanation. Herrick made the statement that we will soon all be cardiophobes if we keep up talking about it and the danger of it. Would you add to your very able discussion the warning to the doctors to be a little more circumspect about what they say?

Dr. Ray E. Logan (closing): I think what the doctor has just said is certainly worth while: surely there is a world of truth in it. A lot of the psychic unbalance of heart disease in all ages is produced by the doctor himself. A doctor hears a murmur. He says, "You have a heart leakage." A doctor in Detroit saw one of his patients going into a bank, and he said, "Are you walking up to the bank?" He then told him, "I told you your heart has a leak, and you are likely to fall

dead any minute." The man actually had a slight roughening of the aorta, due possibly to some mild arteriosclerosis. He had about as much chance of dying of heart disease as I had. He came to me and I explained the whole thing to him, how it was, drew some pictures for him and showed him a diagram of it, and he went about his business and he later told this doctor, "I will be carrying you out feet first."

Here is another thing you all meet with in your practice, since the doctor brought it up. Maybe you have seen it and maybe you have done it. You see an adult or child, and a child especially, who has a cardiac murmur. You don't stop to ask them whether they had rheumatism or not. You don't stop to see whether it is a persistent systolic murmur or not, or whether it is a diastolic. There are all kinds of murmurs you can hear in the heart. We had over six thousand cases; probably made around twenty thousand cardiac examinations, and we were only able to find an average of 471 each year with any suspicion of heart disease at all, and out of that number we had about three hundred and thirty-nine diagnosed as organic rheumatic heart disease. Why? Because the doctors were sending them in with a diagnosis of functional murmurs, and they were telling them that they had an organic heart disease, "a leaky heart." And the minute you tell a patient that he has a leaky heart they are ready to die.

I remember one woman, a high tension, nervous woman. A doctor listened to her heart and told her, "You have a little leakage. I don't think it will do you any damage but don't run around very much." She went home and started to worry about it. So she went to another doctor, and he said, "You go home and go to bed or you will die. You have a terrible lesion." I saw her later. Her home was almost broken up. Her husband was out playing golf at night. I don't know whether they play golf at night, but that's what he told her. They were having a dickens of a time. When she called me over, I asked her to sit up on the bed so I could examine her chest and she said, "I would fall dead because the doctor said so." I asked her to sit on a chair and I practically had to lift her on to the chair. And that was all due to some doctor telling her something that she didn't have, because she didn't have a heart defect at all.

RIEDEL'S STRUMA

C. H. TEARNAN, M. D.

DECATUR, ILL.

Chronic thyroiditis was first described by Riedel in 1896, but he had one recognized case in 1883. It is considered a rare disease. Payr, in 1927, said there were 70 cases on record. Definite records on the number of cases later than that were not found, but there might be as many as two hundred. I wish to add two cases.

Read before the Section on Surgery, Illinois State Medical Society, Springfield, May 15, 1934.

This disease usually occurs between the ages of 30 and 40. Some writers state that it is more common in men, others in women. The majority seem to think it is divided about equally. It apparently is not dependent upon a previous goiter. A typical struma is rapid in growth and pressure symptoms predominate. The disease may last from only a few weeks to six months. Although it is considered inflammatory there is no temperature rise. One or all lobes of the thyroid may be involved. The growth is characterized by extreme hardness,—called iron hardness by Riedel. The size varies and the shape is generally that of the thyroid gland. Grossly the tissue is usually whitish grey, friable and often of striped appearance. The color may be light or reddish brown and yellow.

In 1912 Hashimotos described struma lymphomatosa which Ewing has since classed as an early stage of Riedel's struma. In this type the entire thyroid is not iron hard, but fibrosis is present and perithyroiditis may be found. Grossly such a thyroid is of beefy consistency with fibrous spots. The microscope has followed the progression. There is marked stroma lymphocytic infiltration with later formation of lymphoid follicles. Germ centers may or may not develop. They are composed of lymphoblasts, recticular cells, plasma cells and mitotic figures. As time goes on the whole gland becomes practically a solid mass of lymphoid tissue poor in blood vessels. Connective tissue replaces the cellular infiltration and finally the few remaining acini are surrounded by a solid mass of connective tissue. The lymphoid types are friable while the connective tissue types are tough.

In some cases reported eosinophile cells were dispersed in the connective tissues. The latter occasionally showed hyaline degeneration. On the whole the blood vessels are few and those present show inflammation throughout. Hyaline degeneration and obliteration of the blood vessels has been noted. The inflammation spreads and involves the fasciae and muscles so there is great rigidity. The larnyx, trachea, large blood vessels and nerves are involved in the infiltration by connective tissue. Despite the inflammation there is no suppuration. The mass can be moved as a whole, but each lobe cannot be mobilized separately. The carotid pulsations appear to be weaker than normal. Because of the hardness and pressure symptoms the vast majority of

cases have been diagnosed as carcinoma, and microscopic diagnosis of carcinoma has even been made. In carcinoma of the thyroid, the gland is usually nodular while here the enlargement is usually smooth and one is unable to determine a definite boundary. In this struma the overlying skin is intact and the lymph glands are not involved. Dysphagia is far more suggestive of malignancy than thyroiditis. A distinction must also be made between other inflammatory changes; actinomycosis, tuberculosis and syphilis are among them.

Let us assume that Riedel's struma is due to infection and that the source of infection most likely is to be found in the mouth and throat. Streptococci, staphylococci, tubercle bacilli, typhoid bacilli and the spirochaete have all been blamed by various authors. Erkkes and Wegelin observed relations with influenza while Bruengor and Reist attach some importance to overdosing with iodine. I cannot tell you positively which one is or was on the right track or whether there is just reason for considering all these factors. Crane is satisfied that chronic thyroiditis is a distinct disease entity and has no relationship to the inflammation found in exophthalmic goiters, toxic or non-toxic adenomata.

The early cases are frequently without symptoms. One of the early symptoms is dyspnea Naturally restlessness and apprehension follow. Dyspnea may be an early symptom even in an early case and the growth does not necessarily have to be large. Occasionally there are mild toxic symptoms without exophthalmos or bruit. The basal rate is not high and may be minus. Do not be misled by a high basal in the face of other evidence. Frequently the thyroid is tender with pains radiating up the neck. Sometimes the pains are described as drawing pains. Hoarseness due to recurrent laryngeal involvement and dyspnea from tracheal pressure progressively increase. Roentgenograms should show a compressed trachea in the later stages. Some of the rings may be destroyed.

Operation is indicated to remove the diseased gland and to relieve the grip of the iron claw. The operation is difficult because of the adhesions, immobility, hemorrhage, failure to find lines of cleavage and frequently distortion of the anatomical layout. Pulling on the growth increases the dyspnea. If possible, remove all the diseased tissue. At least remove or sever the isthmus, and take out wedges from the lateral lobes if they cannot be removed. Crile's muscle decompression might help in a severe case. Tetany may be encountered post-operatively as a clean sweep should be made if possible. Parathormone intramuscularly and calcium intravenously should then be used. The operation should be followed up by deep x-ray therapy. Myxedema will usually be found after operation and x-ray and it will be necessary to supply thyroid extract. All foci of infection found should be cleared up unless that was done pre-operatively. Particularly look after the teeth and nasopharnyx.

Cures have been reported even when all of the mass was not removed. How can we tell when we have a cure? How many years must elapse? There are not many cases on record in this country and I wonder just how much follow up has been undertaken and for what period of years. The disease is almost certainly fatal if not cared for. A mortality of 12% was reported in 1932. I believe that covered only the operative deaths. Complete recovery is no doubt possible, but we know of frequent recurrences and would be very slow to give assurance of a cure particularly when recurrence has been recorded twenty years after operation.

Riedel's struma must be considered in every thyroid case where a tumor is present or suspected. The time worn but not outworn plea for earlier diagnosis is made.

I must admit that the first case to be reported here was diagnosed as carcinoma. I have some distinguished company. I console myself a trifle because a short time before I had removed a smooth carcinoma of the thyroid which was very hard and involved all lobes and produced rigidity.

Case 1. Female. Examined April, 1933. Married. Age forty-eight years. Family and past history essentially negative. Had noted thyroid enlargement for about six months. Symptoms were dyspnea, hoarseness, choking. All symptoms had been progressing for three weeks before examination and apprehension was marked. There had been only slight loss of weight. All lobes of the thyroid gland were enlarged, but the left lobe was twice the size of the right. The lobes were smooth and of extreme hardness. Diagnosis of carcinoma was made and operation advised, feeling that we might be able to remove the growth and follow with deep x-ray therapy. At least we could do a decompression and relieve some of the pressure symptoms. Operation, April, 1933. Usual collar incision was made. The anterior muscles were found adherent and the large mass on the left side was adherent. The muscles and fasciae were involved. After some

dissection an attempt was made to get under the mass for ligation. At that point a mass the size of a baseball literally snapped off. Hemorrhage was controlled and as much more tissue as was removed was found rigidly encasing the great vessels. Another smaller mass was noted high in the neck. The ribbon muscles had been split and were not sutured. The removal of the one mass appeared to release the trachea somewhat. The patient made a good recovery so that she was able to get downtown. Two x-ray treatments were given, but more were refused as the dyspnea and choking became worse at that time. Hoarseness increased to brassiness as did the respiration. The voice dropped to a whisper. The struggle for breath was terrific. I have never seen a patient in her right mind who was as panicky as that poor woman. Tracheotomy was performed sixteen days after the first operation. The trachea was found pushed or pulled three fingers breadth to the right and the left jugular was practically in the midline. There was undoubtedly a rapid continuation of the process which was raging before operation or an acute process became engrafted on the old. It is also possible that the phrenics became paralyzed or that the process had become substernal and tracheal collapse occurred below the tracheotomy tube as death came three days after the second operation. Autopsy was refused.

Microscopic Report: There are only small foci of recognizable thyroid tissue. Most of the tissue is made up of a great variety of cells, most of them large, sometimes forming giant cells, but always showing transition to fibroblasts. Among these small irregular thyroid alveoli are scattered. There are also abundant lymphocytes and plasma cells. There are a few mitoses, yet there is no definite evidence of malignancy.

Diagnosis: Chronic Ligneous Thyroiditis (Riedel's Struma).

Case 2. Female. Examined July, 1933. Unmarried. Age 36 years. Family history essentially negative. Past history: Several operations; tonsillectomy, hysterectomy, appendectomy for ruptured appendix, intestinal obstruction. Present illness: In May, 1933, there was sudden onset of sorethroat, swelling of thyroid, pains in head and mastoid region, weakness, nervousness, irritability and loss of weight. Basal metabolic rate was plus 36. Operation in June at a distant clinic where an operative diagnosis of carcinoma of the thyroid gland was made. The patient stated that all of the gland was removed and the tissue reported not malignant. In July the patient presented masses of iron hardness on both sides, which appeared fixed to the muscles. Hoarseness, tightness, and dyspnea all present. Patient apprehensive the same as Case I. A diagnosis of Riedel's struma was made and re-examination of the tissue microscopically bore out that diagnosis. Very few thyroid alveoli, little or no colloid, follicles with germinal centers, marked increase in connective tissue, and excess of lymphoid tissue sets this case down quite definitely, I believe, as one of Riedel's struma. The patient was already taking thyroid extract and was advised to continue with its use. X-ray therapy was started the day after examination. In all there were eighteen treatments. By August, 1933, there was a remarkable decrease in the size and hardness of the masses. All pressure symptoms were still present but in much lessened degree. In September the larynx became mobile for the first time and there appeared to be no muscle involvement. The masses were steadily decreasing in size. Basal metabolic rate in October was minus 3. By December there was an apparent cure. Patient started work in February and now reports no return of growth and no symptoms referable to her Riedel's struma.

Millikin Building.

DISCUSSION

Dr. L. E. Bovik, Waukegan, Ill.: Dr. Tearnan has undoubtedly thoroughly covered the literature on this rare and interesting disease. There is very little to add to what he has said. However, in reviewing the literature myself it seems that the consensus of opinion is that the disease is more prevalent in the female than in the male; that is, probably twice as frequent in the female as in the male.

The symptoms that call for intervention, of course, are the dyspnea and the resulting panicky or nervous condition from this dyspnea. These are the most marked symptoms that the patient presents. Some of these patients have a rise in metabolic rate, althogh we encounter cases in which the metabolic rate apparently remains normal. This factor of metabolism must be taken into consideration after operation for the removal of this iron hard mass, for not infrequently do we have following it the symptoms of myxedema.

As to whether radiation is useful in these cases, there is a difference of opinion among men who have treated them. I believe the largest group of cases that have been reported by any one man was by Crane, who in 1931 reported twenty-eight cases, and in the study of this group he did not believe radiation of any value.

Very interesting is the fact that cases have been reported in which a very definite diagnosis of ligneous struma was made and in which the patient refused any type of treatment. Some of these cases were followed up, and on return for observation after a period of time, there has been a spontaneous cure evolved.

Riedel's struma is a very interesting pathological condition in the study of the thyroid disease, and on which until just very lately, such a small amount of work has been done. Lately, however, it has been given more attention, and its differential diagnosis has been more clearly established.

It is my belief that with care a differential diagnosis can be made between Riedel's struma and thyroid malignancy. Dr. Tearnan in his paper has covered the essential points of differential diagnosis of thyroid malignancy and this interesting clinical condition of the thyroid very well.

A differential diagnosis is of prime importance to the patient because in malignancy the prognosis is distinctly bad whereas with surgical intervention in Riedel's struma the patient can experience relief and probably a complete cure; at least, the alleviation of all disquieting symptoms.

Dr. Lindon Seed, Chicago: I wish to present my experience with Riedel's struma which differs somewhat from what I have been taught or read. I have operated on six cases of Riedel's struma, of these three had the typical history of Dr. Tearnan's second patient; that is, swelling of the thyroid, tenderness and soreness in the neck, and a slight fever in the afternoon, 99 to 100, which later returned to normal. Of these patients, one woman had a malignant hypertension with nephritis, in addition to the Riedel's struma. No matter what was done she was obviously going to die. We had an excellent opportunity to find out what would happen to a Riedel's struma. I did a biopsy on the right lobe, the left lobe was exactly of the same appearance as the right lobe and was left alone. She died three months later, at which time I obtained the left lobe at the autopsy. Strange to relate a microscopic section of the left side showed much less fibrosis and lymphocytic infiltration and many more thyroid follicles. Apparently, with the passage of time there was a marked tendency to spontaneous recovery.

In view of this experience, in the next four cases I saw I advised no treatment whatever. Two of them were nurses and one of them was a doctor, all have been followed for one to three years with a complete spontaneous recovery. I lost track of the fourth case. A month ago I operated on another patient in whom the disease had affected only one lobe. The physician thought it was a carcinoma, and there was some justification for this opinion. It was a typical Riedel's struma. We removed a very small piece of the gland, the symptoms entirely disappeared, and the patient became well. In most instances it does not require removal of a very large piece to remove the soreness, tenderness, and the progressive enlargement.

Although it is the common opinion that all Riedel's struma should be operated on, I have made up my mind, that unless the condition is producing symptoms of obstruction, as in Dr. Tearnan's patients, I would leave it alone.

Dr. Tearman (in closing): As to the first case, I want to say the diagnosis, had we not thought it was carcinoma, would probably have been easier. I want to emphasize the presence of the iron hardness. I don't think you will ever experience anything in the thyroid as hard as that. Another thing, in carcinoma of the thyroid, you usually find nodules. Our experience with Riedel's struma at least has been that the mass is smooth; it isn't nodular like it is in carcinoma or the adenomatous type of goiter.

The second case was one that had been operated on elsewhere some time previous to coming to see us. A diagnosis of malignancy had been made. She presented herself there with symptoms of dyspnea and the iron hardness in the thyroid region. We made the diagnosis of Riedel's struma and asked for a recheck on the slides. The recheck diagnosis was Riedel's struma. This patient was put on x-ray treatment and, in all fairness to Dr. Bovik, who discussed the paper, we felt that the x-ray in that case was the one thing that saved her life, because up to that time she was losing ground. Whispering voice and choking were present. She had all of the pressure symptoms. After only a few x-ray treatments she made quite a remarkable advance and, at the end of four or five months was perfectly well and able to return to work.

It may be possible that in some of these cases, just as Dr. Seed said, unless they have pressure symptoms, they will get well spontaneously. However, we would not feel secure as yet in making a positive diagnosis of Riedel's struma in early cases without operation and microscopic diagnosis. Other diseases of the thyroid can closely resemble a Riedel's struma, and may be the ones that cure spontaneously. In the types where you have a large mass that is shoving the trachea off to one side, you might say in almost an S-curve, where there is a lot of pressure, where the vessels and nerves are involved, as in the first case, we feel that they should be operated upon, and then give a course of x-ray treatment afterwards.

This disease is rather rare and, as time goes on, we may have to change our views.

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BILATERAL EMPYEMA COMPLICATING BILATERAL LOBAR PNEUMONIA IN THE LAST TRIMESTER OF PREGNANCY

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This case is reported because a thorough search of the literature has failed to reveal a single similar case report. It is known that bilateral pneumonia is not common, bilateral empyema is still more rare, and such a combination occurring in

From the Surgical Service of Dr. Harry Jackson, Cook County, Hospital, Chicago.

pregnancy is probably very rare. Cases such as this have no doubt occurred but have not been reported. Therefore, we believe this to be the first reported case, and especially interesting is that the patient appears to be on the road to recovery.

CASE REPORT

No. 1327787: A white female, M. S., aged 18 years, was brought to the examining room of the Cook County Hospital on December 8, 1932, with a note from Dr. Frederick A. Lofton who stated the patient has "pneumonia and pregnancy." She was admitted to the obstetrical service of Dr. Louis Rudolph, where she gave a history of a seven month pregnancy. The previous day she had had a chill followed by pain in the chest later in the day; and the coughing up of reddish brown material at night. The chest pain started in the left upper anterior portion and later became generalized over the entire chest.

In the past she had had measles, mumps, chickenpox, whooping cough and diphtheria as a child. Her menses started at the age of twelve years, were of three days duration, with a twenty-eight day interval. Her last menstrual period had ended April 2, 1932. This was her first pregnancy.

On examination she appeared acutely ill, orthopneic, with cyanosis of the lips and finger-tips, and constantly groaned as she held her hands to ease the pain in her left chest. Her temperature was 98° F, pulse 124, and respiration 36 per minute. The blood pressure was 114/80. The pharynx was markedly reddened. Her breasts were engorged. The borders of the heart were R H B 3 cm, and L H B 8 cm. The cardiac tones were soft and weak, and the rate was regular. There was impaired resonance with numerous moist crepitant rales over the right lower lobe posteriorly. Over the left lower lobe there was impaired resonance, bronchial breathing and bronchophomy posteriorly. The abdomen revealed the fundus of the uterus 22 cm. above the symphysis pubis. The fetus was in the R O P position, and increased fetal movements were noted. The F H T was 160 per minute counted in the right lower abdominal quadrant. Dr. H. F. Oakes made the diagnosis of a bilateral lower lobe pneumonia with a normal seven month pregnancy, which was confirmed by Dr. Helen Button. The patient was given glucose intravenously, fluids, and morphine for rest.

Labor: The following day the temperature was 102°, pulse 140 and respiration 54. At 4 A. M. patient began to have labor pains. Her general condition appeared improved. Labor proceeded uneventfully until 10:45 A. M., when she spontaneously delivered a still-born premature infant. Her condition became very serious following the delivery and she was placed in an oxygen tent.

Course: For three days she remained in the oxygen tent and with relief of the severe anoxemia, her condition improved somewhat. On December 12, 1932, she had consolidation of the entire right lower lobe.

On December 14, 1932, she became irrational and remained so for the next four days. She also developed a papular erythema which became generalized over the entire body. Her temperature stayed at 102°. Pulse varied from 96 to 130, and respirations between 30 and 40 for the next few days.

On December 26, 1932, when she appeared to be doing very well her temperature went up to 103°, pulse increased to 140 and respiration to 40. She had a dry, hacking cough, and examination of the chest revealed flatness and diminution of breath sounds over the right lower chest. The left lower chest showed impaired resonance, bronchial breathing and coarse rales. A thoracentesis was performed on the right chest and thick purulent material was obtained. She was transferred to the surgical service of Dr. Harry Jackson on December 28, 1933.

Laboratory: On December 8, 1932, the day of admission the W B C was 4,100 with 78% polys. The following day the W B C was 7,100 with 88% polys. On December 12, 1932, the W B C was 15,100 with 85% polys.

X-ray of the chest on December 27, 1933, revealed bilateral occlusion of the lower two-thirds of each lung field.

Surgical Course: The left chest was aspirated on December 29, 1932, and this also revealed thick purulent material. Culture of this material gave a mixed growth of hemolyzing streptococci and staphylococcus albus. Both chests were then aspirated alternately for the next six days. She was scheduled for a rib resection on January 6, 1933, but her condition became very grave. Closed drainage was therefore instituted in the left chest, using the trocar method. After withdrawal of 400 cc. of pus, her condition improved.

Drainage continued well until January 9, 1933, when the tube in the left chest slipped out. Her breathing was much impaired and anoxemia became very evident. Another tube was re-inserted and she was again placed in an oxygen tent. She remained twenty-four hours in the tent. Her condition improved considerably so that on January 13, 1933, a closed drainage was also instituted in the right chest.

For the next three days nothing unusual happened. On January 16, 1933, the left tube again slipped out of place; also, the right tube became clogged and stopped functioning. Her respiratory rate rapidly increased and marked cyanosis became very evident. Bilateral closed drainage was therefore re-instituted and about 400 cc. of pus was withdrawn from both pleural cavities. Then everything again went well for a few days.

On January 20, 1933, she became completely disorientated due to a marked psychosis. In this state she pulled both tubes out of her chest and both had to be re-inserted. Fluoroscopy, after the psychosis subsided, revealed fluid in the left chest to the level of the 3rd rib with air also present, and a fluid level at the 8th rib on the right side. Her condition gradually improved as both pleural cavities drained freely until January 26, 1933. At that time it was noted that she had multiple abscesses in the right thigh and right arm. Within the following few days about ten abscesses were incised and drained over the thighs, arms, and lumbar region. With drainage from all the abscesses and both pleural

cavities, her condition became very slowly a little better.

In the entire month of February, it was necessary to re-insert the closed drainage tubes three times on the left side and twice on the right side. In addition other abscesses were incised in the lumbar region, the arms and legs, about eight in all. At the end of the month, just when there appeared to be some improvement, she developed another complication that was the worst one of the entire group. All of her large joints, the shoulders, elbows, wrists, hips, knees and ankles became red, tender, very painful and swollen. This brought on the pressure sores on the back as she could be moved only with great pain.

On March 6, 1933, a bilateral footdrop with complete paralysis of both lower extremities, for no obvious reason but the cachexia, was noted. In this desperate condition the patient appeared to be gradually going downhill. On March 20, 1933, when her condition was as low as it possibly could be, she insisted that she felt fine and must be allowed to go home. This was taken up with the family and her mother removed her from the hospital by ambulance on March 28, 1933. At discharge, her condition was very poor. She was very emaciated, drained from about ten different places where the abscesses had been incised; she had a paralysis of both lower extremities, and multiple pressure areas on the back.

Subsequent Course: The patient was entirely dismissed from our minds until one day one of us (N. F.) received a call to see this patient. She was seen on October 22, 1933, at her home and again on December 3. Her condition had improved considerably. All of the abscesses had ceased draining, the pressure areas had cleared up; only the bilateral foot-drop was still present. There was a small sinus draining in the right chest, but otherwise nothing else. Although the patient is far from recovery, she appeared very much better than when she left the hospital. This was undoubtedly due to her mother's excellent care.

SUMMARY

A case of bilateral empyema complicating a bilateral lobar pneumonia in the last trimester of pregnancy is reported for the first time. In addition she had a pyemia with multiple abscess formation, an infectious polyarthritis, and a bilateral foot-drop. After a spontaneous delivery her condition was always poor. Bilateral closed drainage was instituted and multiple abscesses were incised. For several days she had a marked psychosis. When her condition was very poor her mother took her home to care for her. When last seen on December 3, 1933, the patient was an invalid, but appeared to have a good chance for recovery.

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GOLDSODIUMTHIOSULPHATE IN THE TREATMENT OF PULMONARY TUBERCULOSIS

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Based upon experimental observations tending to show that the clinical use of gold derivatives produced an inhibitory effect on the growth of tubercle bacilli and appeared to manifest a certain attraction for tuberculous tissue, clinicians have from time to time centered their efforts upon the use of this metal in the treatment of pulmonary and other forms of tuberculosis.

From the time of Robert Koch and his experimental work with gold cyanide in 1890 until Moellgard's striking reports, the many attempts at the therapeutic application of various gold salts in tuberculosis met with an unfavorable response, chiefly as a result of the dangerous reactions which followed their use. Moellgard's favorable reports on the experimental and clinical use of Sanocrysin revived interest in the possibilities of gold therapy. The severe reactions occurring in connection with the dosage he suggested served to demonstrate, however, that the remedy was not devoid of the objectionable qualities of previously recommended gold preparations. The tendency to discard Moellgard's salt based upon the failure to duplicate his results experimentally was in a measure prevented by many optimistic reports of its use clinically.

Following a study of the available literature upon the subject the administration of goldsodiumthiosulphate was decided upon in a series of sanitorium patients.

Dosage. No definite agreement exists with regard to the dosage of goldsodiumthiosulphate. Danish clinicians recommend massive doses; an initial dose of 250 to 500 mg. and this amount increased until 1500 mg. is administered at a dose. Numerous case reports testify to brilliant results following the use of such radical dosage. More recent reports, however, indicate the advisability of conservatism in relation to dosage. An initial dose of 25 to 50 mg. progressively in-

creased according to the patient's tolerance constitutes the safest plan of treatment.

The chemical is given intravenously dissolved in from 10 to 20 c.c. of sterile distilled water. A total of 6 to 8 grams is advised over a period of from twelve to fifteen weeks.

In our series of cases we commenced with a minimum dose of from 10 to 40 mg., increasing the amount given by 10 mg. at weekly intervals until the patient developed an unfavorable reaction. This was regarded as the "reaction point." The next dose was lowered to the amount tolerated and following one or two such doses in the absence of unfavorable manifestations the "reaction point" dose was again given. If the reaction did not recur the dosage was increased by 10 mg. every week until a maximum of 250 mg. was reached. A total of fifteen maximum doses were administered at weekly intervals. In a few subsequent cases the initial dose was 50 mg. and succeeding doses were increased by 50 mg. Patients unable to tolerate 250 mg. were given their greatest tolerance dose at intervals of one week.

The patient should be confined to bed for at least twenty-four hours following the intravenous injection.

Types of patients treated. Cases selected for treatment included for the most part far advanced types with positive sputum, whose pulmonary involvement did not permit of the application of collapse therapy or in whom collapse therapy failed to produce the desired result.

The presence of the following conditions were considered as contraindications in the administration of goldsodiumthiosulphate:

1. Albuminuria; hematuria; pyuria.
2. Hypertension; arteriosclerosis.
3. Hypertrophic emphysema with cyanosis.
4. Gastrointestinal complications.
5. Amyloidosis; diabetes mellitus.

CASE REPORTS

Case 1. E. V. Aged 29 years. Onset with pleurisy in 1924; admitted to sanitorium, December 21, 1928. Lung findings on admission; fibrocaseous tuberculosis both upper lobes; cavitation right upper lobe; no serious complications. Temperature 98.6 to 100.2; pulse 88 to 112; weight 90 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Treatment started January 10, 1931, with initial dose of 10 mg. Lung findings on January 10, 1931; small areas of cavitation both upper lobes; many moist rales both upper lobes. Temperature 98.6 to 100; pulse 100 to 132; weight 93 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Reactions during treatment; 30 mg. fourth dose caused albuminuria; 30 mg. eighth dose, diarrhea; 60 mg. twelfth dose, erythema; No further reactions until 190 mg. which produced a blotchy erythema. 250 mg. the second dose in this amount followed by albuminuria. 250 mg. given September 17 followed by headache, dizziness, vomiting and heavy albuminuria which persisted until December 16, 1931.

Clinical observations, January, 1932; Temperature lower; pulse slower; constitutional symptoms improved. During September and October, 1931, the maximum afternoon temperature was 99.0 and pulse 80. Lung findings remain practically unchanged. Sputum always positive. Weight 101¾ lbs.

Case 2. S. N. Aged 26 years. Onset following an acute respiratory infection in 1924; admitted to sanitorium on September 10, 1927. Lung findings on admission; moist rales upper lobe of right lung; moist rales lower lobe left lung; artificial pneumothorax for a short period prior to his admission; phrenicexeresis right side, June, 1929. Temperature 98.6 to 99.0; pulse 74 to 90; weight 139 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis far advanced B.

Treatment started, January 10, with initial dose of 10 mg. Lung findings on January 10, 1931; Relaxation right lung about 40% result of phrenic operation; moist rales throughout the right lung; moist rales scattered throughout left lung; slight cyanosis; Temperature 98.6 to 99.2; pulse 84 to 110; weight 159; Sputum positive. Diagnosis: Pulmonary tuberculosis far advanced B.

Reactions during treatment; 160 mg. caused dizziness; 230 mg. produced a slight erythema; 250 mg. given once a week for twelve weeks with no reaction; 250 mg. the 15th dose was followed by chills, fever, vomiting and severe prostration. Treatment discontinued, October 15, 1931.

Clinical observations November, 1931: Temperature 98.4 to 99.8; pulse 80 to 90; symptoms unchanged. Lung findings the same as on January 10. Sputum positive; weight 160 lbs.

Case 3. B. L. Aged 36 years. Onset insidious, 1918; admitted May 12, 1929. Lung findings on admission; right spontaneous pneumothorax; fluid removed and pneumothorax maintained for a brief period then discontinued owing to progressive adhesion formation; right phrenicectomy, May 1930; few moist rales left apex. Temperature 98.0 to 100.4; pulse 90 to 96; weight 90 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis far advanced B.

Treatment commenced February 26, 1931, with dose of 30 mg. Lung findings on this date; right hydropneumothorax; moist rales left apex. Temperature 97.6 to 100.8; pulse 80 to 96; weight 95 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Reactions during treatment; 70 mg. followed by severe headache and malaise; 100 mg. caused stomatitis; 250 mg. sixth dose in this amount produced nausea, vomiting and dizziness; 200 mg. given a week later re-

sulted in a severe constitutional reaction; treatment discontinued November 15, 1931.

Clinical observations November and December, 1931: Temperature normal; pulse 84 to 90; condition appears improved. Lung findings essentially the same. Sputum positive; weight 99 lbs.

Case 4. S. B. Aged 40 years. Onset with chronic cough in 1929; admitted July 31, 1930. Lung findings on admission; moist rales upper lobes both lungs; x-ray shows a small cavaity in the right apex. Temperature 98.6 to 99.8; pulse 80 to 86; weight 102 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis far advanced B.

Treatment started on March 15, 1931 with dose of 40 mg. Lung findings on this date the same as when she was admitted. Temperature 98.6 to 1008; pulse 78 to 84; weight 119 lbs.; sputum spositive. Diagnosis; Pulmonary tuberculosis far advanced B.

Reactions during treatment; 160 mg. first reaction produced burning sensation in the throat of two days duration; 250 mg. on September 17, the fifth dose in this amount caused albuminuria which was still marked on October 29; treatment was stopped.

Clinical observations November 1931; Maximum temperature 99.8; maximum pulse 92; clinically improved. Lung findings; decreased moisture right upper lobe. Sputum positive; weight 131 lbs.

Case 5. A. S. Aged 36 years. Onset with hemoptysis in 1930; admitted to the sanitorium March 29, 1931. Lung findings on admission; moist rales both upper lobes; cavity left apex; dry pleurisy over left lower lobe. Temperature 97.0 to 98.6; pulse 70 to 86; weight 152 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis far advanced A.

Treatment commenced August 7, 1931, with dose of 25 mg. Lung findings on this date the same as upon admission. Temperature 98.6; pulse 74 to 100; weight 163 lbs. Sputum negative; positive until April 12, 1931. Diagnosis; Pulmonary tuberculosis, far advanced A.

Reactions during treatment; 250 mg. fifth dose in this amount caused severe headache, nausea, vomiting, diarrhea and prostration. Treatment discontinued.

Clinical observations December, 1931; Temperature normal; pulse normal; weight 162. Pulmonary findings remain unchanged; sputum negative. General condition improved; coughs less; expectorates less.

Case 6. S. S. Aged 24 years. Onset after acute respiratory infection in 1923; admitted March 5, 1930. Lung findings on admission; moist rales with signs of cavitation upper lobe of the left lung; tuberculosis infiltration right apex. Temperature 100 to 103; pulse 100 to 110; weight 172 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, moderately advanced C.

Treatment commenced January 10, 1931 with dose of 10 mg. Lung findings on the above date; left hydropneumothorax complicating therapeutic pneumothorax given on September 10, 1930 for severe hemoptysis; few moist rales right apex and upper lobe. Temperature 100.2 to 103; pulse 100 to 128; weight 146 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced C.

Reactions during treatment; 70 mg. caused mild albuminuria; 240 mg. produced "sores" in the mouth; 250 mg. followed by stomatitis; injections stopped for three weeks; 250 mg. the seventeenth dose in this amount with no reaction on December 2, 1931.

Clinical observations December, 1931; Maximum fever 100; maximum pulse 124; weight 164 lbs. Left lung expanded; no fluid; no cavitation; pneumothorax discontinued because of adhesion formation; few moist rales right apex. Sputum positive. June 1, 1932; sputum negative since March 1932; weight 184 lbs.; maximum temperature 100; maximum pulse 110; condition improved.

Case 7. I. B. Aged 40 years. Onset with gastrointestinal symptoms in 1919; admitted September 18, 1930. Lung findings on admission; fibroulcerative tuberculosis involving the upper two thirds of the right lung and upper half of the left lung; cavitation right apex. Temperature 97 to 99; pulse 90 to 104; weight 155 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Treatment started January 10, 1931 with initial dose of 10 mg. Lung findings on this date practically the same as upon admission; cough a troublesome symptom. Temperature 98 to 99; pulse 76 to 90; weight 175 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Reactions during treatment; none. 250 mg. administered on June 18, 1931; patient went home.

Clinical observations, June 20, 1931; Temperature normal past month; pulse 70 to 90; weight 182 lbs. Lung findings unchanged; sputum positive; "feels better"; cough improved.

Case 8. A. C. Aged 20 years. Onset with cough and blood streaked sputum in October, 1929; Admitted June 9, 1930. Lung findings on admission; moist rales left upper lobe with signs of cavitation; bronchovesicular breath sounds right upper lobe with moist rales after cough. Temperature 98.6 to 99.6; pulse 80 to 90; weight 137 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Treatment commenced August 27, 1931; initial dose 50 mg. Lung findings on this date practically the same as upon admission. Temperature 98.6 to 100; pulse 76 to 100; weight 150 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Reactions during treatment; 250 mg. the third dose in this amount followed by a severe reaction; further treatment refused, October 21, 1931.

Clinical observations November, 1931; Temperature normal; pulse 80 to 96; weight 147 lbs. Lung findings unchanged; sputum positive.

Case 9. H. S. Aged 45 years. Onset catarrhal in 1925; admitted July 17, 1930. Lung findings on admission; moist rales with signs of cavitation upper lobe of the right lung; most rales upper lobe left lung. Temperature 97 to 100; pulse 92 to 100; weight 115 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Treatment commenced on January 10, 1931; initial dose 10 mg. Lung findings on this date, essentially the

same as on admission. Temperatur 98.6 to 100; pulse 96 to 110; weight 115 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Reactions during treatment; 80 mg.; nausea; abdominal distress; 100 mg. followed by severe gastro-intestinal upset; 110 mg. injected one month later produced a marked unfavorable reaction; further treatment discontinued April 15, 1931.

Clinical observation May, 1931. Pulmonary involvement rapidly progressive. General condition bad.

Case 10. R. S. Aged 40 years. Insidious onset, 1926; admitted July 23, 1930. Lung findings on admission; fibrocaseous tuberculosis upper half of left lung and upper third of right lung; small cavities upper lobes. Temperature 98 to 100.6; pulse 88 to 100; weight 104 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Treatment commenced January 10, 1931; initial dose 10 mg. Lung findings on this date same as on admission. Temperature 98.6 to 100.6; pulse 80 to 106; weight 108 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Reactions during treatment; No reaction until 200 mg. which was followed by nausea and vomiting. Treatment discontinued with maximum dose of 220 mg. on June 3, 1931 because of severity of reactions.

Clinical observations; during April, 1931, maximum pulse rate was 96; maximum temperature 99.4; weight 113 lbs.; in May she developed an acute exacerbation with a maximum temperature of 103. Following this the trend of her progress was unfavorable. Sputum positive at all times.

Case 11. A. H. Onset with hempotysis in 1924; admitted May 17, 1929. Lung findings on admission; hydropneumothorax left chest complicating artificial pneumothorax which was maintained until December, 1929; small cavity right upper lobe. Temperature to 100.4; pulse to 124; weight 104 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Treatment commenced on January 10, 1931, with 10 mg. Lung findings on this date; large cavity left upper lobe; thickened pleura over left lower lobe; moist rales upper third right lung; small cavity right apex. Temperature 97.8 to 98.6; pulse 80 to 90; weight 110. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Reactions during treatment; 20 mg. caused diarrhea; 30 mg. produced a transient albuminuria; 80 mg. followed by erythema; 90 mg. the fourteenth dose, no reaction; 120 mg. given on May 7, 1931, resulted in a severe reaction with an exacerbation of his cough and bloody expectoration. Further treatment refused.

Clinical observations; during May, 1931, his temperature ranged from 97.8 to 99.8; pulse 88 to 116; weight 119 lbs. Clinical findings indicative of improvement in right lung; rales much less numerous; left lung the same; thoracoplasty left side; patient succumbed.

Case 12. J. S. Aged 28 years. Onset with cough and hoarseness July, 1927; admitted June 26, 1929. Lung findings on admission; fibrocaseous involvement upper two thirds of both lungs; asthmatic bronchitis; tuberculosis laryngitis. Temperature 98.6; pudse 80 to 94; weight 167 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Treatment commenced January 10, 1931; initial dose 10 mg. Lung findings on this date the same as on admission. Temperature, pulse and weight the same as on admission. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Reactions during treatment; 20 mg. followed by albuminuria; no further reactions; 250 mg. the eighteenth dose in this amount administered on December 2, 1931.

Clinical observations December, 1931; Local and constitutional findings remain unchanged. Temperature and pulse the same; weight 173 lbs. Sputum positive. February 10, 1932; developed bronchopneumonia; died three days later.

Case 13. H. D. Aged 15 years. Onset with catarrhal symptoms April, 1929; tuberculosis contact; admitted June 10, 1929. Lung findings on admission; extensive involvement right lung; large cavity right upper lobe; fine moist rales left auxillary region. Temperature 98.6 to 100.4; pulse 84 to 100; weight 86 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced B.

Treatment commenced February 26, 1931; initial dose 10 mg. Lung findings on this date the same as on admission; artificial pneumothorax tried right side with no success owing to adhesions. Temperature 97 to 98.6; pulse 96 to 104; weight 105 lbs. Sputum positive. Diagnosis; Pulmonary tuberculosis, far advanced A.

Reactions during treatment; none; 100 mg. injected on May 6, 1931, was the last dose given on account of the patient's unfavorable progress.

Clinical observations May, 1931; Temperature to 99.4; pulse to 100; weight 103 lbs. X-ray reveals three areas of cavitation in the left lung. Sputum positive.

Summary of cases presented. The cases discussed are representative of the types of patients treated and afford an insight into the problems involved in the administration of the salt and the evaluation of its effect on the clinical course of the disease. The difficulty in evaluating the therapeutic response to any form of chemotherapy in the light of the remissions and exacerbations of the average case of pulmonary tuberculosis is at once apparent. The tendency to ascribe the favorable phase of the clinical cycle of the disease to a remedy administered coincident with this period should not be overlooked. With regard for these considerations we have endeavored to summarize the salient points relating to our observations.

Toxic Reactions. Toxic reactions of goldsodiumthiosulphate may be mild, moderate or severe. They have been grouped as follows: Skin; erythematous patches; generalized erythema. Mucous membrane; superficial ulcerations of the

SUMMARY OF CASES TREATED

Case	Period Treated	Total Grams of "Gold"	Temp.	Pulse	Weight	Sympt.	Physical Findings	X-ray	Sputum	Remarks
1.	1-10-31 to 9-17-31	3.5	Impr.	Impr.	Plus 8 lbs.	Impr.	No definite change	Same	Remains positive	Symptoms improved
2.	1-10-31 to 10-15-31	6.5	Not impr.	Impr.	Plus 1 lb.	No change	No change	Same	Remains positive	Gained 20 lbs. before treatment
3.	2-26-31 to 11-15-31	3.4	Impr.	Impr.	Plus 4 lbs.	Impr.	No change	Same	Remains positive	Symptoms improved
4.	3-15-31 to 10-29-31	2.8	Impr.	Same	Plus 12 lbs.	Impr.	Impr.	Not impr.	Remains positive	Gained 17 lbs. before treatment
5.	8-7-31 to 11-27-31	2.3	Always Normal	Same	Same	Impr.	Same	Same	Remains negative	Gained 11 lbs. before treatment
6.	1-10-31 to 12-2-31	5.0	Impr.	Same	Plus 38 lbs.	Impr.	Impr.	Impr.	Remains positive	Lost 26 lbs. before treatment
7.	1-10-31 to 6-18-31	3.0	Impr.	Same	Plus 7 lbs.	Impr.	Same	Same	Remains positive	Gained 19 lbs. before treatment
8.	8-27-31 to 10-21-31	1.2	Impr.	Impr.	Lost 3 lbs.	Impr.	Same	Same	Remains positive	Gained 13 lbs. before treatment
9.	1-10-31 to 4-15-31	.63	Not impr.	Not impr.	Loss in weight	Worse	Worse	Worse	Remains positive	Gained 18 lbs. before treatment
10.	1-10-31 to 6-3-31	2.4	Impr.	Impr.	Plus 5 lbs.	Same	Same	Same	Remains positive	Acute exacerbation during treatment
11.	1-10-31 to 5-7-31	.65	Not impr.	Not impr.	Plus 9 lbs.	Impr.	Impr.	Impr.	Remains positive	Succumbed to thoracoplasty
12.	1-10-31 to 12-2-31	7.1	Always normal	Same	Plus 7 lbs.	Same	Same	Same	Remains positive	Died of bronchopneumonia 2-10-32
13.	2-26-31 to 5-6-31	.42	Worse	Same	Lost 2 lbs.	Not impr.	Worse	Worse	Remains positive	Gained 18 lbs. before treatment

mouth; stomatitis. Gastrointestinal; anorexia; nausea; vomiting; diarrhea. Fever. Shock.

The toxic manifestations are apparently due to metallic poisoning. Mild reactions are usually transitory and of minor clinical consequence. In most patients a progressive increase in tolerance can be established. Many patients who developed mild or moderate reactions with minimal doses failed to show any signs of toxicity with increased dosage. The occurrence of a slight rise in temperature, a transient gastrointestinal upset, a few erythematous patches on the skin or a few superficial ulcerations on the mucous membrane of the mouth may not require a decrease in dosage. Generalized erythema, prolonged or severe alimentary disorders, persistent albuminuria, high fever or signs of shock call for extreme caution in the administration of the salt. Treatment should be discontinued until the unfavorable symptoms subside and subsequent amounts of the remedy given materially reduced. In some cases it may be advisable to stop the treatment entirely.

Comment. An analysis of our clinical observations must in a measure be rather speculative in nature. The following deductions with reference to the effect of the salt upon the clinical condition of the patients reported are worthy of mention.

The remedy appeared to have no influence upon the presence of tubercle bacilli in the sputum. Positive cases remained positive. The only exception to this observation was Case 6 whose sputum became negative about three months after the injections were discontinued.

Physical and x-ray findings were not definitely influenced in seven patients. Some improvement incident and subsequent to treatment was appar-

ent in three cases. Unfavorable progress of the pulmonary involvement was noted in three instances.

Symptomatic improvement, at times striking, was observed in eight patients under treatment. In two cases no definite symptomatic change was evident. In three patients the clinical course was decidedly unfavorable. Two of the latter were extremely intolerant to the salt.

Conclusions. Based on the clinical observations in our series of cases one may be justified in the assumption that goldsodiumthiosulphate has failed to definitely prove its therapeutic efficiency in the treatment of pulmonary tuberculosis. The favorable response in some of the patients, however, would seem to indicate that the remedy is not entirely devoid of therapeutic merit and that its cautious administration is perhaps worthy of consideration in selected cases.

TREATMENT OF UTERINE FIBROIDS

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Treatment of fibroids of the uterus at the Michael Reese Hospital has undergone a definite change during the past eleven years. During this period which ended January 1, 1934, 1,001 private and service patients were treated.

A survey of the types of treatment used in this series of 1,001 patients shows that supravaginal hysterectomy was performed 663 times, vaginal hysterectomy 79 times, and total hysterectomy 49 times. 137 patients were treated by myomectomy; 15 of these were by the vaginal route and the other 122 by laparotomy. The remaining 73 patients were treated by radium.

A study of the frequency (66.3%) with which supravaginal hysterectomy was used throughout this eleven year period shows that this operation is the method of choice in the treatment of uterine fibroids. Furthermore, the frequency of its use remained constant. Supravaginal hysterectomy is the simplest and the most rapid of the various major operations. This procedure should be practically bloodless; there is no risk of bladder injury if the vesical reflexion of the peritoneum is carefully stripped off the anterior surface of the uterus; neither is there danger of ureteral involvement except in the occasional patient in whom the fibroid is either intraligamentary or aberrant.

With the abdomen opened it is possible to inspect and palpate the tubes and ovaries, and to perform such surgical procedures as are indicated by the pathology found. Likewise the general abdominal cavity may be explored, and appendectomy may be performed.

This procedure removes the tumor-bearing corpus uteri, thus obviating the recurrence of fibroids. It leaves the cervix intact and is the operation of choice when the cervix is healthy or amenable to treatment. The advantages of leaving the cervix in situ are first that the cervix serves as an anchorage for the vaginal vault and second that the endocervix provides the necessary vaginal moisture, thus preventing dyspareunia.

In hysterectomy of any type vaginal preparation is as essential as abdominal. Ethylene oxygen anesthesia has proven entirely safe and satisfactory and is used routinely. Ether is added only occasionally at the moment of opening the abdomen and, if necessary, for beginning closure. The clamp method of removal and the ligation method each have their advocates. There is no outstanding advantage to either technique except that in removal of markedly eccentric and large fibroids ligation sometimes facilitates the procedure.

The presence of the cervical stump which is cut across in supravaginal hysterectomy has been blamed for much of the morbidity in this operation. To obviate the potential infection from the endocervix, coning out, cauterization, carbolization and iodinization have all been used. After experience with each of these procedures it has been found equally safe to omit all of them, resorting only to a transverse wedge shaped resection of the supravaginal portion of the uterus. Suture closure of the cervix is the first step following removal of the tumor bearing corpus, after which there is no further risk of contamination. Each uterine artery should be ligated separately.

It is desirable to implant the cut ends of the round ligaments together with the suspensory ligaments of the ovaries into the lateral margins

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of the cervical stump. Thereafter the vesical peritoneum is utilized for complete peritonealization.

TREND IN TYPES OF TREATMENT

	Total	1923	1933
Myomectomy	12.2	21.9	7.6
Vaginal myomectomy	1.5	....	....
Supra vaginal hysterectomy	66.3	56.2	57.6
Total hysterectomy	4.9	1.4	14.6
Vaginal hysterectomy	7.9	5.5	18.1
Radium	7.3	15.0	2.1

The incidence of total hysterectomy for the entire series was 4.9%. However, this operation was performed 10 times as frequently in 1933 (14.6%) as in 1923 (1.4%). It is indicated whenever the cervix requires removal and whenever vaginal hysterectomy is contraindicated. Total hysterectomy requires a most accurate knowledge of normal and abnormal pelvic anatomy; the possibility of injury of ureters and bladder is greater than in supravaginal hysterectomy. For this reason total hysterectomy demands a higher degree of operative skill. When all these conditions are fulfilled, the morbidity and mortality should be no higher than that following supravaginal hysterectomy.

Those who recommend total hysterectomy as a routine procedure do so on the basis that carcinoma of the cervical stump is a real menace. It is of interest to note that such a carcinoma is not known to have developed during this eleven year period. In this operation the first step should be suture closure of the cervix vaginally to obviate the risk of peritoneal contamination from the cervix when the latter is withdrawn through the abdominal incision. The Richardson technique has proven most satisfactory among the various methods recommended for total hysterectomy. It is quick, safe and comparatively bloodless. The vaginal vault is closed without drainage and the round ligaments and the uterosacral ligaments are attached to it. If median approximation of the round ligaments causes too much tension one or both should be peritonealized in situ, otherwise these patients complain of pain in the groins which persists until the round ligaments pull out of their artificial attachments.

Vaginal hysterectomy was performed in 7.9% of the entire series. This operation also shows a marked increase in frequency during this period. In 1923 the incidence was 5.5% and by 1933 it had jumped to 18.1%. This operation has many distinct advantages which in competent hands should make it the operation of second choice. The abdominal cavity is scarcely invaded, nevertheless the adnexae may be inspected and removed if necessary, the diseased cervix is removed and cystocele or rectocele or both can be corrected at the same time without undue prolongation of the operation and in the same operative field. The convalescence should be simple and uncomplicated provided there is careful asepsis and hemostasis. There is practically no trauma to peritoneum or bowel. This results in a definitely lowered incidence of shock, ileus and peritonitis.

Vaginal hysterectomy should be employed only in those instances in which the tumor is not too large to be removed intact. Morcellation or bisection of the uterus may be used in those tumors which are too large to be delivered intact through the vaginal canal but these two procedures should be avoided if possible because of the increased risk from trauma or infection. It is preferable to remove larger tumors by the abdominal route. Vaginal hysterectomy is also contraindicated when the uterus is fixed, when there is parametrial infiltration, when there is palpable adnexal pathology or when there have been previous pelvic abdominal operations.

In this operation, as in abdominal types of hysterectomy, either the clamp method or the ligation method may be used. The ligation method is simple with a freely movable uterus and a roomy vagina. The clamp method is safer if the broad ligament attachments and fundal attachments of the uterus are not readily accessible. The clamp method should be done stepwise, each bite of the clamp being ligated in turn.

The fundal attachments are sometimes more easily approached by turning the corpus backward into the posterior peritoneal opening via the cul-de-sac. The prolapse of abdominal content into the vaginal vault can be prevented by a temporary pack or by lowering the head of the table. Closure without drainage is routine. In closure the broad ligaments are approximated and sometimes imbricated. Their upper margins, together with the round ligaments, are attached to the sub-pubic ligaments to forestall herniation of the bladder.

Myomectomy is primarily the operation of choice in those women who choose to maintain their fecundity. It is also indicated for simple pedunculated tumors, single infected tumors,

aberrant fibroids and for similar types of fibroids in association with pregnancy. Vaginal myomectomy (1.5% in this series) may be used for cervical fibroids and for submucous fibroids. It is of special value in the latter type when they have been expelled from the uterine cavity.

It is ill advised to combine a vaginal myomectomy and an abdominal hysterectomy. The attachments of the vaginal fibroid may be infected or sloughing. The ligated stump should be given not less than six weeks to heal before the abdominal operation is performed.

Abdominal myomectomy was performed in 122 (12.2%) of the entire series; 81 of these women were under 35 years of age. The incidence of this operation in 1923 was 21.9%. This decreased to 7.6% in 1933 because of the increased morbidity and mortality due chiefly to postoperative infection.

Radium was employed in the remaining 7.3% of the patients. The decrease in the use of radium from 15.0% in 1923 to 2.1% in 1933 is explainable by the many contraindications to the use of radium in the treatment of uterine fibroids together with the comparatively high percentage of failure following its use. The generally accepted contraindications to radium are:

1. *Size.* Radium is contraindicated for fibroids larger than the size of a 12-14 week pregnancy.
2. *Position.* Subserous or pedunculated submucous fibroids.
3. Tumors producing pressure or pain symptoms.
4. Tumors undergoing degeneration or infection.
5. Rapidly growing tumors.
6. Tumors associated with inflammatory pelvic disease, postoperative adhesions or other new growths such as endometriosis.
7. Stenosis of the cervix.
8. Severe bleeding or marked anemia.
9. Pregnancy.
10. Retrodisplacement.
11. *Age.* The severity of the symptoms of the post radiation menopause is in direct relation to the prematurity of the induced menopause. Thirty-five years was the original arbitrary age limit under which radium was contraindicated. General experience has raised this level to 40 years. In this series 24.3% of women were still menstruating at age 46. For this reason it seems desirable to consider radium, if at all, only in the fifth decade and as close to the expected menopause as can be determined.
12. It is further to be noted that radium is contraindicated after the menopause is established.
13. Radiophobia.

Even though the use of radium is the safest procedure it should not receive primary consideration in the treatment of fibroids because of the high incidence of failure (11.11% in this series). It also has certain other definite disadvantages; chief among these is the inability to inspect and palpate the pelvic and abdominal organs.

The total mortality in this series was 0.7% (seven deaths). The causes of death were pulmonary embolism twice, cerebral embolism once, peritonitis three times (two streptococcus and one Welch bacillus) and intestinal obstruction once. Of these 7 patients 5 had supravaginal hysterectomies and 2 myomectomies, a mortality for myomectomy of 1.64% while by contrast the mortality among 791 patients in whom every type of hysterectomy was done was only 0.63%.

The noteworthy postoperative complications which occurred in this series are listed below.

POST-OPERATIVE COMPLICATIONS

	Morbidity	Mortality
Wound infections	19	(2)
Peritonitis	1	3
Eviscerations	6	1
Wound resuture—No infection	2	..
Intestinal obstruction	2	(1)
Thrombophlebitis	14	(1)
Embolism	..	3
Febrile 10+days	7	..
Pneumonia	6	..
Shock	5	..
Cardiac	4	..
Hemorrhage	2	..
Psychosis	2	..
Ant. tibial paralysis	1	..
Recto-vaginal fistula	1	..
Total	72—7.2%	7 deaths—0.7%

It should be noted that carcinoma of the body of the uterus was found four times (0.4%) and sarcoma five times (0.5%), an incidence of malignancy of only 0.9%.

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DISCUSSION

Dr. Edwin J. DeCosta, Chicago: Several interesting and correlated problems have been analyzed in the examination of the case records of these 1000 patients. Perhaps the most interesting is that associated with the question of the etiology of fibroids.

Although the true histogenesis of fibroids is not known it is generally accepted from the work of Meyer and Becher that uterine fibroids do not arise from embryonic rests, nor metaplasia, nor from the walls of blood vessels, but rather from an abnormal growth of hitherto normal myometrium. Fibroids possess certain attributes that are lacking in most other neoplasms. For example, they usually occur during the child-bearing years and may retrogress after natural or artificial cessation of ovarian function. Thus it has not seemed

unreasonable to assume that secretion of the ovaries is intimately associated with myomagenesis.

The present tendency in the search for the origin of fibroids seeks the cause in some dysfunction of those hormones that regulate the growth and physiology of the pelvic organs. There is a glaring discrepancy in this theory. During puberty and gestation, peaks in hormonal activity, there is no increase in myoma formation. Likewise fibroids that derive their nourishment from other sources than the uterine blood vessels, as through vascular adhesions, may continue to grow after the menopause. Witherspoon, and Witherspoon and Butler have recently demonstrated what appeared to them to be a clearcut interrelation between the presence of ovarian follicle cysts, hyperplasia of the endometrium and fibromyoma. Their interpretation is as follows: 1. Follicular cyst formation produces a hyperestrinism; 2. the unopposed action of estrin in the absence of the corpus luteum secretion produces a hyperplasia of the endometrium, and 3. this same action of estrin on the myometrium, if sufficiently prolonged, results in fibromyomatous growths. Concerning the origin of the follicle cysts; in white women a general glandular upset, with special emphasis on the anterior pituitary influence, is considered most important, while in colored women a disturbed blood supply resulting from chronic pelvic inflammation leads to follicle cysts.

Analysis of adnexal pathology occurring in association with fibroids in this series does not substantiate the conclusion of Witherspoon. For instance, follicle cyst formation, the primary basis of this theory, was observed in only 244 patients, 26.1 per cent while corpora lutea were noted 129 times, 13.7 per cent. The presence of the latter theoretically would interfere with the action of estrin and thereby prevent fibroid formation. Hyperplasia of the endometrium and polypoid growths occurred in 177 patients, and it is of particular interest, that only 34.4 per cent of these presented follicle cysts. Of 72 patients in whom the endometrium was considered normal, 38.9 per cent had follicle cysts. Concerning evidence of pelvic inflammation, salpingitis occurred in 19.9 per cent, while Witherspoon's observations reach 97.6 per cent.

ABNORMALITIES IN THE POSITION AND FORM OF THE VERMIFORM APPENDIX

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The frequency of appendicitis and the large number of surgical operations performed for this condition make it essential that both physicians and surgeons be acquainted with congenital anomalies in number, position, location and form of the vermiform appendix.

In 1906, I reported upon the gross abnormalities of the appendix discovered in 3,550 autopsies performed in the Cook County Hospital, Chicago, between 1893 and 1905, and the reader is referred to that paper for a number of interesting conditions discovered many of which were of pathological origin. In the present paper, I am surveying the more recent general literature in regard to a few of the more commonly reported anomalies which, although in no sense of frequent occurrence, yet may happen to occur in any particular case and therefore should be borne in mind by practitioners.

Development of the Processus Vermiformis. Embryologically, the vermiform appendix is merely the terminal portion of the cecal pouch. From the developmental standpoint, there may be a number of anomalies: The cecum itself may fail to develop in which case, of course, the appendix would also be congenitally absent; there may be a rudimentary cecum without the appendix; the cecum, including the appendix, may develop in an abnormal position in the peritoneal cavity but be otherwise normal; there may be full development of the cecal part of the general cecal pouch but failure or only a rudiment of the appendicular part; finally, there may be normal development of the cecum and its appendicular part but failure of differentiation between the true cecal sac and the appendix. In addition, as we shall see, the appendix is occasionally duplicated, the so-called "double" appendix and, more frequently, there is the "giant" appendix which may result from a congenital malformation or from a pathological condition occurring *in utero* or after birth.

Differentiation between the cecum proper and the appendix appears to take place in two stages. A primary stage is stated to occur early in embryonal life when a distinction can be made between a larger proximal portion of the general cecal pouch, the cecum proper, and a smaller distal portion, the future appendix. The second stage occurs about the time of birth when the distinction between the portions becomes quite clearly marked.

Normally, the appendix is found free in the right iliac fossa and is about 6 to 12 cm. in length, the average being about 8 cm. Its normal situation is on the posteromedian aspect of the cecum with the tip pointing downward.

Anomalies in Position of the Appendix. Wakely made careful observations on the position of the

appendix in more than 5,000 cases as seen in the operating, post-mortem and dissecting rooms. Omitting the influence of inflammation and neoplastic disease in altering the normal position of the appendix, he gives the following general statistical values:

In 5,000 Cases

Position of the Appendix	Per Cent.
1. Anterior or pre-ileal	0.94
2. Splenic or post-ileal	0.5
3. Pelvic, on psoas muscle, near or hanging over the brim of the pelvis	32.11
4. Sub-cecal, beneath the caput ceci	2.02
5. Post-cecal and retrocolic	64.38
6. Ectopic positions	0.04

In the first position, the tip of the appendix is directed upward and forward over the terminal part of the ileum. In the second position, the appendix lies in the ileocecal fossa under cover of the terminal part of the ileum. The tip of the appendix in the third position is usually directed downward on the psoas muscle. In the sub-cecal position, the appendix with its mesentery is usually twisted in a clockwise direction from left to right and its tip is usually directed upward.

Wakely thinks that the high percentage of retrocolic positions is in accordance with the general experience of surgeons and anatomists but differs markedly from the statistics published by previous writers and from those given in text-books. The retrocecal type of appendix may be found: (a) free in a post-cecal or retrocolic pouch of peritoneum; (b) held in contact with the cecum or ascending colon by a short mesentery; (c) adherent to the cecum or colon which with the appendix forms the anterior wall of a retrocolic pouch of peritoneum; (d) behind the cecum and ascending colon and partly or entirely extraperitoneal.

When one is unable to locate the appendix in the usual way, the posterior surface of the cecum should be explored to see if the apendix is retrocecal. Spivak. A freely movable terminal part of the ileum does not necessarily mean that there is not a retrocecal appendix, but a terminal part of the ileum bound down by peritoneal folds to the psoas muscle does suggest a retrocecal appendix.

In regard to Wakely's figures for retrocolic positions, different authors and text books give percentages varying from 18 to 38 for retrocecal appendix. Operative statistics are usually double the anatomic statistics, but in the former, it should be remembered that pathological conditions in the vicinity may play an important role.

An appendix located in any of the atypical positions mentioned, may give rise to a wrong diagnosis; particularly in the case of women, an ovaritis or salpingitis may be thought of when the appendiceal pain is not localized in the usual site. As McKinnon mentions, inability to correctly locate the position of the appendix is responsible for the still high mortality of appendicitis whether operated on or not.

Absence of the Appendix. Congenital absence of the appendix — sometimes it is an agenesia rather than a true absence. It has been noted in museum specimens, at the autopsy table, at the dissecting table and at operation. Kelly and Hurdon in their text-book say: "Cases of complete absence of the appendix have been described but it is to be doubted that such observations have always been accurate. Nevertheless there are a few authentic cases." In my report of 1906, previously mentioned, in which the findings in 3,550 autopsies were referred to, there was not one case of congenital absence of the appendix noted.

Bradley states that in 8,102 cases of examinations in the autopsy or dissecting room in which the presence or absence of the appendix was noted, only 2 showed congenital absence. Thus, as a clinical entity, congenital absence of the appendix, has owing to its rarity very little significance. The first reference to the subject is traced to Morgagni in 1719. It was reported by Wm. Hunter in 1762, by V. Haller in 1765, and several apparently authentic cases have been reported since.

Dorland and Bradley and Spivack have presented good analytical statistical studies on this subject in 1925, 1929 and 1931 respectively. Dorland collected 37 cases from the literature which he regarded as authentic cases of agenesia of the appendix; Bradley added 4 more but states that some of the earlier cases included in Dorland's list are not accepted by some critics as authentic. Spivack, including 2 personal cases, increased the number of reported cases to 44. Of these, nine were discovered at the autopsy table, 6 on the dissecting table, 4 are museum specimens and the others were found at operation. Spivack considers that some of the cases are not true agenesia or congenital but rather cases of hypoplasia and expresses the opinion that the cases of true agen-

esia or congenital absence of the appendix scarcely amount to one-half. In many cases, a very careful examination will reveal some vestige of the organ either as a very slight papular elevation externally or as a hollow depression corresponding to the orifice of the appendix on the mucosal side.

Jacobs, in 1928, reported a case of laparotomy which disclosed a bicornuate uterus with one cervix and an elongated cecum with no adhesions and without any trace of an appendix or mesoappendix.

It is quite evident that a complete and thorough autopsical or dissecting room report is necessary in order to establish a case of true absence of the appendix. The mere fact that at operation an appendix was not found, even following a more or less extensive search, does not establish an authentic case. Such for instance, is the case reported by Maurer in which it is stated that an operation was undertaken on a diagnosis of appendicitis but no appendix was found. In Marie's case, the cecum presented no trace of an ileocecal appendix. The malformation was congenital.

Duplication of the Appendix. Like absence of the appendix, its duplication, the so-called "double" appendix, is an anatomical rarity. Most text-books either do not mention this anomaly or else say that the reported cases are not authenticated. Double appendix is so infrequently seen that it presents very little practical interest.

In my report on 3,550 autopsies in which peculiarities of the appendix were particularly noted, there was no case of duplication. Many surgeons of wide-reputed experience have never seen a human cecum with two appendices. One must be most careful not to mistake a Meckel's diverticulum or other formation for a supernumerary appendix. Young, in 1911, operated on a case of double appendicitis; the two appendixes contained pus, one was ruptured. These two appendices, each had a separate mesoappendix, had their bases about three centimeters apart of each other. In 1929, Braatz reported the case of a young woman who in 1909 had had her appendix removed for appendicitis; 10 months later, similar symptoms appeared and at a second operation, another true appendix pathologically affected was found in the vicinity of the usual site in the cecum. It was histologically verified as regards structure. There was no doubt that an appendix had also been removed at the first operation. Elwyn reported a case of forked appendix, there was a normal limb from the right cecal sacculation, another limb from the left; the two branches being fused at the distal end. In Goldschmidt's case, the appendix was also removed at operation. Between the site of the appendix and the ileal inlet, and fixed in the anterior wall of the cecum, a nut-sized structure covered with omentum and with a mesentery was found. Histological examination showed that both the appendix and the removed structure were exactly the same in their elements, i. e., two appendixes and both gave rise to appendical attacks in the patient. In 1931, Walthard reported the finding of a double appendix associated with an umbilical fistula. A duplicate appendix was present in the umbilical protusion. Both appendices arose from a common base and the author suggests it may have been a case of an appendix bent double upon itself. Lettau, in 1903, had reported an almost exactly similar case. In 1932, Berthold reported a case in a woman 60 years old in whom operation disclosed 2 appendices. One was 7 cm. and the other 5 cm. long; the first was in its normal site and the second about 2 to 3 cm. lateral to it. Both were histologically verified as appendices. Recently Clavel and Colson report a case of double appendix discovered in the course of a laparotomy. On opening the abdomen, the appendix was found to be retrocecal; a second appendix was found about 1 cm. distant from the first, in a double-barreled gun position. The second was located internally to the first. The two appendices were about the same length and each had an independent cecal implantation. The authors mention a similar case seen by Routier except that the two appendices were united at the distal end. Pratt reports a case of double appendix associated with imperforate anus and other congenital anomalies found at autopsy in an infant 11 hours old. Each appendix had its own mesenteric attachment and the two were on opposite sides of what apparently was the cecum. Rasmussen has reported a case of double appendix associated with bicornuate uterus.

Giant Appendix. The so-called giant appendix may be due to massive hypertrophy of the organ but it is more usually a cystic dilatation. In a general way, an enormous appendix may result from inflammation, infection, cecal distension or

from a malignant tumor. Infection need not necessarily be present.

Virchow was the first to describe a large cystic appendix, in 1863, under the name of pseudo-mucinous cyst. Haimant and Mathieu reporting in 1923, estimated at about 183, the number of cases of giant apendix to be found in the literature. This included 3 cases personally observed by them. The condition is found most frequently between the ages of 20 and 30 years. It is due, apparently, in most cases, to the retention of products of secretion. These cysts may rupture, may become infected, may undergo malignant neoplastic change.

The largest cystic appendix reported was removed by Neumann at operation upon a man aged 69 years. The cyst was the size of an adult's head. Kelly reported a case in a woman of 60 who came to the hospital to be treated for metrorrhagia; she had never had any digestive trouble nor the least abdominal painful crisis; she was operated upon for fibroid uterus and in the course of the operation the appendix shaped like a banana was found to be 30 cm. in length and 15 cm. in circumference. An appendectomy and a hysterectomy were performed. The patient recovered. Forgue, Mourgue-Molines and Lapeyre report a case in which the appendix after removal measured 13 cm. in length and 12 cm. in circumference. In its original state in the body, it was vastly distended and its walls were thin and transparent. Larget and Lamare reported a case of cystic appendix 10 cm. long and 4 to 5 cm. thick which had given no symptoms. It was a surprise finding during an abdominal operation.

The contents of these cysts may vary; sometimes being gelatinous and sometimes mucoid or pseudo-myxomatous, which gives rise to the so-called appendicular mucocele; sometimes the contents are clearly fluid, either aqueous or serous—the hydro-appendix. The cause is usually some degenerative process or a cellular hyper-secretion. Several authors have associated hydro-appendix with a localized tuberculosis.

In the case reported by Newell, Campbell and Frere the removed appendix weighed one pound six ounces. Its diameter where it came off from the cecum was five centimeters. The condition apparently resulted from a chronic inflammation; part of the removed mass was fibrous. Matassov reported a case of fatal ileus from strangulation of intestine by an appendix 13 cm. long.

Ectopic Appendix. A word should be said about the rare condition of ectopic appendix, not secondary to some pathological process. In Wakely's 5,000 appendicular examinations, in only 2 was the appendix found ectopic. In one, it was prehepatic while in the other it was found lying free with the cecum below the stomach and transverse colon. In Hickman's case, the appendix was in a diverticulum rather low in the right inguinal fossa. Buisson's case of subhepatic ceco-appendicular ectopia was diagnosed roentgenologically and similarly in the case of ectopia reported by Santoro. In Keaton's case, the cecum and the appendix were on the left side, with the ascending colon running parallel to the transverse colon. Appendix has been found in the left side of the abdominal cavity, it has been found in right sided and in left sided inguinal hernial sacs, and also in umbilical hernial sacs. Abnormal position, angulation, fixation, constriction, and abnormal location of the appendix, secondary to pathological anatomic changes are as common as congenital anomalies of position and location are rare.

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THE HIPPOCRATIC OATH

I swear by Apollo the physician and Aesculapius and health and all healing and all the gods and goddesses that according to my ability and judgment I will keep this oath and this stipulation. To reckon him who taught me this art equally dear to me as my parents, to share my substance with him and relieve his necessities if required. To look upon his offspring in the same footing as my own brothers and to teach them this art if they shall wish to learn it without fee or stipulation; and that by precept, lecture and every other mode of instruction I will impart a knowledge of the art to my own sons and those of my teachers and to disciples bound by a stipulation and oath according to the law of medicine but to none others. I will follow that system of regimen which according to my ability and judgment I consider for the benefit of my patients and abstain from whatever is deleterious and mischievous. I will give no deadly medicine to any one if asked nor suggest any such counsel and in like manner I will not give a woman a pessary to produce abortion. With purity and with holiness I will pass my life and practice my art. I will not cut persons laboring under the stone, but will leave this to be done by men who are practitioners of this work. Into whatsoever houses I enter I will go into them for the benefit of the sick and will abstain from every voluntary act of mischief and corruption and further from the seduction of males or females, of freemen or slaves. Whatever in connection with my professional practice or not in connection with it I see or hear in the life of men which ought not to be spoken of abroad I will not divulge, as reckoning that all should be kept secret. While I continue to keep this oath unviolated may it be granted to me to enjoy life and the practice of the art, respected by all men in all times; but should I trespass and violate this oath may the reverse be my lot.

MADE NO DIFFERENCE

"Is your son improving his opportunities in college?"

"Yes; he's just as tough on $1,000 a year as the millionaire's son in the next room who spends $5,000."

ALPHA-DINITROPHENOL AND DINITRO-O-CRESOL, AS STIMULATORS OF METABOLISM

Actions and uses of dinitrophenol. Promising metabolic applications. W. C. Cuttings, H. G. Mehrtens and M. L. Tainter (Division of Neuropsychiatry and the Department of Pharmacology, Stanford University School of Medicine, San Francisco, California), J. A. M. A. 161:193-195 (July 15, 1933) No. 3.

Alpha-dinitrophenol is a yellow crystalline solid, slightly soluble in water, more soluble in alcohol or ether. Solutions may be made up to 3% in water by heating with the addition of one-half the weight of sodium bicarbonate. Dinitrophenol causes an increase in metabolism within one minute after injection into experimental animals, or somewhat more slowly when given gastrically. With doses of 10 mg./kg. or less, the increase in metabolism is about 50%, with larger doses, metabolism may be increased fourfold or more, until the animal produces heat so fast that it may be killed by the resulting fever. Return of metabolism to normal levels may require 6-48 hours, depending on the dose and the route of administration. The increase in metabolism is due to a direct stimulation of cellular metabolism and is not accompanied by an increase in muscular activity, nor by important changes in the circulatory system unless asphyxia results from inadequate oxygenation of the blood, when there may be acceleration of the pulse and rise of blood pressure. There is a marked increase in respiration; the respiratory quotient decreases. Thyroid and suprarenal glands are not indispensable to the metabolic response, since their removal does not interfere with the drug's actions. In diabetic dogs, dinitrophenol has a greatly increased toxicity. Repeated administrations of the drug for from 2-3 months to dogs in doses just short of the fatal, have not resulted in significant injury to important organs, as shown by studies of the urine, icteric index, van den Bergh test, organs at autopsy and tissues microscopically. In 8 human cases, single doses of 3-5 mg./kg. body weight orally increased the basal metabolic rate from 20-30% in the first hour and maintained it at that level for about 24 hours, when it gradually fell, reaching normal levels on the third day. None of the patients experienced or showed signs of nervousness, anxiety, trembling, hunger or palpitation. Given orally or subcutaneously these doses caused no changes in temperature, respiration or pulse rate after 2 months of daily administration. Single doses from 5-10 mg./kg. caused no change in temperature, pulse or respiration, but caused the patients to sweat copiously. Three administrations of single doses of more than 10 mg./kg. gave increases in temperature of three degrees C., or more, in respiration of 15-30 per minute, and of pulse 20-30 per minute all commensurate with the pyrexia. The latter doses are too dangerous for routine use. Daily oral doses of 3-5 mg./kg. were given to 9 patients for from 1-10 weeks. In 6 of these patients, the metabolism was determined repeatedly and found to be maintained at an average of 40% above the initial level. After discontinuance of the drug the rate fell to normal on the third or fourth day. All 9 patients lost body weight

without dietary restrictions: the maximum total loss in any patient was 9 kg. (20 pounds) in 10 weeks. The greatest rate of loss was 1.7 kg. (3-7/10 pounds) a week after a daily dose of 3 mg./kg. The average loss for the entire group was 0.9 kg. (2 pounds) a week; the patients did not suffer from deleterious symptoms as the result of this reducing treatment. The authors point out that there are limitations to and possible dangers from the use of the drug clinically and urge that dinitrophenol be administered under strictly controlled conditions since: (1) the drug was not in these experiments administered continuously for periods longer than three months and not at all to patients with serious disease; (2) the drug manifests increased toxicity in diabetic dogs; (3) excessive doses may cause a fatal hyperpyrexia; and (4) there may exist a possible idiosyncrasy to the drug. Further study is proposed in the treatment of obesity, hypothyroidism and similar depressed metabolic states.

Alpha-dinitrophenol; preliminary report of the Council on Pharmacy and Chemistry of the American Medical Association, J. A. M. A. 101:210 (July 15, 1933) No. 3.

Dinitro-o-cresol as a stimulator of metabolism. E. C. Dodds and W. J. Pope, (Courtauld Institute of Biochemistry, Middlesex Hospital, and the Department of Chemistry, University of Cambridge.) Lancet 2:352-353 (August 12, 1933) No. 7.

It would appear from experimental results that dinitro-o-cresol is more suitable for clinical use than alpha-dinitrophenol as a stimulator of metabolism. It produces the same effects as the dinitrophenol in about ⅓ the amount; toxicity studies indicate that the two compounds are of the same order, and both are active when administered by mouth. The compounds were compared on guinea pigs, using a modification of the Krogh technique.

Toxicity of alpha-dinitrophenol. Report of a case. H. H. Anderson, A. C. Reed and G. A. Emerson. (Laboratory of A. C. Reed and the Pharmacological Laboratory, University of California Medical School), J. A. M. A. 101:1053-1055 (September 30, 1933) No. 14.

Alpha-dinitrophenol was administered to 14 patients for the treatment of obesity. Thirteen of the cases, treated with about 3 mg./kg. daily of the drug for periods up to two months, did not suffer appreciable ill effects. The average weight loss for the first month of therapy was 2.3 kg., and for the second month, 2.1 kg. The diet was limited to foods low in sugar and fat. In the fourteenth patient a case of "qualitative idiosyncrasy" was encountered, with extremely toxic manifestations. This patient had suffered, previous to the administration of alpha-dinitrophenol, from arthritis of the chronic hypertrophic type, and should not have been given the drug, as it is known that individuals with chronic rheumatism, alcoholism, tuberculosis and renal and hepatic disease have a lessened resistance to the agent. It is suggested that Derrien's test (described in detail) be used in determining the presence of alpha-dinitrophenol in the urine of patients under treatment as a means of detecting intolerance to the drug, although in the case of allergy the test of course is of no value. Ninety fasting normal rats were injected intraperitoneally with 10-50 mg./kg. in a single dose. A chart is given showing the lethal range for the drug in the group of animals studied. Fifty per cent of the animals died with 40 mg./kg. and all rats given 50 mg./kg. died immediately after the height of the pyrexia, which occurred within the first hour. No gross evidence of edema or other tissue damage was observed in any animal. Literature on the toxicity of alpha-dinitrophenol is reviewed. The authors urge that the dosage in humans be strictly and conservatively controlled, as it has not yet been demonstrated that the administration of the drug is without danger.

Miscellaneous actions of dinitrophenol: Repeated administrations, antidotes, fatal doses, antiseptic tests and actions of some isomers. M. L. Tainter and W. C. Cutting (Department of Pharmacology and the Division of Neuropsychiatry of the Department of Medicine, Stanford University School of Medicine, San Francisco, California), J. Pharmacol. 49:187-208 (October, 1933) No. 2; through J. A. M. A. 102:405 (1933) No. 5.

Tainter and Cutting observed that repeated administration, by different routes, of alpha-dinitrophenol (1-2-4) to dogs at intervals of three or more days does not result in tolerance within a period of from two to three months. Studies of the urine, van der Bergh tests and icteric indexes all failed to reveal any evidence of a toxic action during such administrations. There were no significant pathologic alterations in the important organs, aside from some possible injury to the spleen. The fatal doses per kilogram for 50 per cent mortality were: rats, 25 mg. subcutaneously; dogs 22 mg. subcutaneously, 20 mg. intramuscularly, 30 mg. intravenously and between 20 and 30 mg. orally; rabbits, 30 mg. subcutaneously, and pigeons, 7 mg. intramuscularly. Excised intestinal strips from rabbits showed response to dinitrophenol in high concentrations only, which depressed the muscle directly. Dinitrophenol may increase the rate and depth of respiration in rabbits previously depressed by toxic doses of morphine, chloral, alcohol or barbital, as does caffeine. The animals may still die, in spite of maintenance of adequate pulmonary ventilation. When the full respiratory stimulation of dinitrophenol has developed, morphine promptly reduces it to normal. Dinitrophenol was not successful in preventing death from just fatal doses of sodium barbital. Arterenol, sodium gluconate, dextrose and insulin, monoiodoacetic acid, quinine and salicylate were found ineffective antidotes in rats. The administration of physiologic solution of sodium chloride or cooling by means of tepid baths exerted a partial antidotal (antipyretic and antimortal) effect. Dinitrophenol failed to prevent death from sodium cyanide in pigeons under conditions in which methylene blue is effective and is of no permanent value as an antidote in cyanide poisoning. No antiseptic action was found against Bacillus coli or

streptococci in vitro, or against trypanosomiasis in rats. Alpha-dinitrophenol (1-2-4) is more effective in producing fever in pigeons, as far as degree of fever and regularity of response are concerned, than are metamononitrophenol and paramononitrophenol, betadinitrophenol or gammadinitrophenol or trinitrophenol. The authors state that these results, taken together with those of their previous reports, establish the basis for the use of dinitrophenol in physiologic and pharmacologic experimentation for increasing metabolism and body temperature. They also indicate the possibility of applying this drug in the treatment of various clinical conditions in which an increase in the basal metabolism, or a fever, might be of benefit. Properly conducted clinical tests are imperative before any clinical applications could be considered.

Toxicity of dinitrophenol. Henry H. Haft (Syracuse, New York), letter to the editor, J. A. M. A. 101:1171-1172 (October 7, 1933) No. 15. A case is reported of a University professor, who although well aware of the possible toxic effects of dinitrophenol, insisted on treatment with the drug for obesity. His weight was 263 pounds (119 kg.); age 39, height 6 feet. He was given 3 mg./kg. of body weight daily in capsule form, and reported daily for examination. For three days no untoward effects were noticed; on the fourth day he complained of a feeling of heat, perspired profusely, complained of pain in the buttocks and down the legs, felt fatigued, and had an uncomfortable feeling in the abdomen. Examination revealed injected conjunctiva, with a definite icteric tint to the eyeballs; beads of perspiration were on his forehead; his pulse rate had increased from 60 to 90; blood pressure was lowered from 125 systolic to 112 systolic; his abdomen was sensitive, particularly over the liver; urine showed a trace of sugar and bile. He stopped the use of the drug, and four days later he felt well again. The icterus had disappeared; the pulse rate was normal; there was no more excessive sweating; and the pain in the buttocks and abdomen had disappeared.

A death from alpha-dinitrophenol poisoning. J. C. Geiger (San Francisco, California), letter to the editor, J. A. M. A. 101:1333 (October 21, 1933) No. 17.

A death is reported resulting from 2.5-5 gm. dinitrophenol. The drug was taken at 11:50 A. M. At 4 P. M. the patient experienced a feeling of apprehension; he became more restless and uneasy, and at 6 o'clock was admitted to the hospital. Restlessness and apprehension increased, with progressive rise in temperature, frequency of pulse rate and respiration, accompanied by profuse perspiration. The temperature rose to 105.4°F. The pulse on admission had been 84; at 9:30 it registered 146 beats a minute. The patient lapsed into delirium and coma at 9:45 and died at 10 o'clock. The rectal temperature at that time was 110°F. plus. Within twenty minutes his body was in boardlike heat rigor.

Use of dinitrophenol in obesity and related conditions. A progress report. M. L. Tainter, A. B. Stockton and W. C. Cutting (Departments of Pharmacology and Medicine, Stanford University School of Medicine, San Francisco, California), J. A. M. A. 101:1472-1475 (November 4, 1933) No. 19.

Results obtained in the treatment of 113 unselected patients (15 males) for obesity (average initial weight 188⅗ pounds) with dinitrophenol or its sodium salt are reported. Capsules containing 100 mg. of the Na salt or 75 mg. dinitrophenol were used; the two forms are therapeutically indistinguishable. One or two capsules were given daily with meals; after a week's interval the dosage was increased until 2-3 pounds of weight loss were produced weekly. No restriction of the diet was made. Patients were examined at intervals of 1-3 weeks. The average length of treatment was 40 days; the longest, 125 days. The average loss of weight was 8⅘ pounds, which with the average duration of treatment of 40 days would give a rate loss of 1½ pounds weekly. It was found that for an average weight loss of 2-3 pounds weekly, 2-9 capsules per patients, or 1.5 capsules for 100 pounds of initial body weight were needed. Data and results obtained in individual cases are given. Of the 113 patients, 7 were lost track of; 19 terminated the treatment for reasons not associated with the drug; 23 were dismissed with treatment completed; 52 were still under treatment; 9 had unpleasant reactions; 3 were uncomplicated failures of the drug. 23.9% of the patients complained of perspiration or a sensation of warmth; 5.3% complained of feeling tired; 3 thought that their nervousness increased; 4 had attacks of dizziness; 3 had gastro-intestinal upsets of brief duration; 6 experienced an alteration of taste sensation; 7% had skin rash with intense itching; in none were there respiratory or circulatory symptoms; hypertension and albuminuria associated with obesity were improved; no evidence of liver damage was found. Results indicate that sufficient dinitrophenol can be used therapeutically to reduce the weight of obese patients 2-3 pounds weekly over extended periods without serious discomfort.

The clinical applications of dinitro-o-cresol. E. C. Dodds and J. D. Robertson (Courtauld Institute of Biochemistry, Middlesex Hospital, London), Lancet 2:1137-1139 (November 18, 1933) No. 21.

Studies on the therapeutic dose of dinitro-o-cresol were made. It was found that a safe dose that will cause a definite increase in basal metabolic rate would appear to lie between 50 and 100 mg. per day for a normal person, or 0.5-1.0 mg./kg. body weight. Under no circumstance should the compound be administered in such quantities as to raise the basal metabolic rate above +50, as grave discomfort and damage will result. Neither the pulse rate nor the blood pressure are of any value in assessing the basal metabolic rate, since the characteristic action of dinitro-o-cresol is an increase in metabolism without a proportionate stimulation of the cardio-vascular system. It is possible by the careful use of the compound to maintain the metabolic rate at 30-50% above normal without the appearance of discomfort or toxic symptoms, and, provided that the diet is not grossly in excess of the normal requirements, the intake can be regulated so that a steady loss of weight

results, without undue privation. It appears that dinitro-o-cresol is in the region of five times as potent as dinitrophenol as judged by clinical results. Toxic symptoms due to excessive doses of dinitro-o-cresol are: sweating, lethargy, severe headache, loss of appetite and a definite pigmentation of the conjunctiva.

The clinical applications of dinitro-o-cresol. II. A study of myxœdema. E. C. Dodds and J. D. Robertson (Courtauld Institute of Biochemistry, Middlesex Hospital, London), Lancet 2:1197-1198 (November 25, 1933) No. 22.

A man, age 42, suffering from myxœdema (and who required 2 grs. daily of thyroid to keep his metabolism at a normal level and rid him of hypothyroidism) was given gelatin coated capsules of dinitro-o-cresol, at the rate of 3 mg./kg. body weight. It was found that dinitro-o-cresol did not alleviate the symptoms of myxœdema despite the fact that the basal metabolic rate was increased. It appears that the relief of myxœdema and the power of raising the basal metabolic rate are two separate functions, one of which is possessed by drugs similar to dinitro-o-cresol but both of which are possessed by thyroxine. It may be also that the increase in metabolism induced by dinitro-o-cresol is different from the normal metabolic process.

ALL IN SAME BOAT

During prom week-end, Dean ——— of the Medical School was rudely awakened by the ringing of his telephone. It was about three in the morning; it must be an important call. Dr. ——— asked drowsily: "What is it?"

The voice said: "Dean we need your help over at our house. We're having a party and Frater Jones has us all worried."

"Well, why call me? Is he seeing elephants and snakes and things?"

"No, Dr. ———, that's why we called. The room is full of them and he can't see any."—*Penn. Punch Bowl.*

Society Proceedings

ADAMS COUNTY

Resolution adopted at a Special Meeting of the Adams County Medical Society, State of Illinois, September 25, 1934:

WHEREAS: It has come to our knowledge that an organization known as the Catholic Women's Maternity Guild has instituted a plan in the City of Quincy and elsewhere which provides for hospital and maternity services for the care of Catholic women, which include pre-natal, partum and post-partum care, at a stipulated sum, which sum is to be paid to the hospital, which in turn will pay the physician for his services, and

WHEREAS: The reasons for this plan, as given, are to counteract the propaganda of birth control, and to make it easier for said expectant mothers to pay for such services, and

WHEREAS: No statistics are now available to prove that Catholic women are being deprived of the privilege of child bearing because of the cost of hospital and medical care prior to and immediately after the period of gestation, and

WHEREAS: Vital statistics show that the birth rate in Quincy is on the increase, and

WHEREAS: The premise upon which this movement is founded is without fact or justification, and

WHEREAS: The "Principles of Medical Ethics" of the American Medical Association provide primarily for the best interests of the sick, and

WHEREAS: Such a plan is in distinct violation of the "Principles of Medical Ethics" of the American Medical Association, and in particular is not in accord with the additional Principles of the Ethics recently adopted at the Cleveland session in 1934, which reads as follows:

First: All features of medical service in any method of medical practice should be under the control of the medical profession. No other body or individual is legally or educationally equipped to exercise such control.

Second: No third party must be permitted to come between the patient and his physician in any medical relation. All responsibility for the character of medical service must be borne by the profession.

Third: Patients must have absolute freedom to choose a legally qualified Doctor of Medicine who will serve them from among all those qualified to practice and who are willing to give service.

Fourth: The method of giving the service must retain a permanent, confidential relation between the patient and a "family physician." This relation must be the fundamental and dominating feature of any system.

Fifth: All medical phases of all institutions involved in the medical service should be considered separately. These institutions are but expansions of the equipment of the physician. He is the only one whom the laws of all nations recognize as competent to use them in the delivery of service. The medical profession alone can determine the adequacy and character of such institutions. Their value depends on their operation according to medical standards.

Sixth: However, the cost of medical service may be distributed, the immediate cost should be borne by the patient, if able to pay, at the time the service is rendered.

Seventh: Medical service must have no connection with any cash benefits.

Eighth: Any form of medical service should include within its scope all qualified physicians of the locality covered by its operation who wish to give service under the conditions established.

Ninth: Systems for the relief of low income classes should be limited strictly to those below the "comfort level" standard of incomes.

Tenth: There should be restrictions by non-medical groups on treatment or prescribing unless formulated and enforced by the organized medical profession.

Therefore Be It Resolved: That any member of the Adams County Medical Society, who in any way encourages, participates, or lends his services to this movement, shall be guilty of unethical practice, and

upon being proven guilty of such violation, by trial, as provided in the Constitution and By-Laws of the Adams County Medical Society will automatically be expelled from membership in this Society.

Be It Further Resolved: That a copy of these resolutions be sent to every member of the Society, to the Secretary and members of the Council of th Illinois State Medical Society, to the Secretary of the American Medical Association, and to the Editor of the Illinois State Medical Journal.

BUREAU COUNTY

RESOLUTION ON DEATH OF DR. CLARENCE EARL SMART

WHEREAS, Doctor Clarence Earl Smart was a man of sterling character; was an honored and respected citizen and member of Society; and,

WHEREAS, He was a faithful and sincere worker in his chosen profession and a member of the Bureau County Medical and of the Illinois State Medical Society.

WHEREAS, He has been called to his long rest.

BE IT RESOLVED, That we members of the Bureau County Medical Society, herein assembled, do offer to the relatives and friends, our most sincere sympathy; that we assure them that our brother physician and associate will be missed by us as the years go by; and,

BE IT FURTHER RESOLVED, That the resolution be spread among the minutes of this Society and that a copy be sent to the ILLINOIS MEDICAL JOURNAL.

CHAMPAIGN COUNTY

On October 11, the Champaign County Medical Society was addressed by Dr. John Wolfer of Northwestern University Medical School. His subject being "Reports and Treatment of Gastric and Duodenal Ulcers. It was something that was handled in a masterful manner, both from the medical and surgical standpoint of treatment.

On November 8, the address of the evening will be by Dr. Francis Eugene Senear, on "Some Common yet Important Dermatological Conditions." That will close the year's work with the exception of election of Officers at the December meeting.

Dr. W. M. Honn, the President for the past year, was recently appointed a member of the Board of the Elks Crippled Childrens Clinic, which meets in Champaign every three months.

Respectfully yours,
W. M. HONN, President.

COOK COUNTY

CHICAGO MEDICAL SOCIETY

Regular Meeting, Wednesday, October 10, 1934
PROGRAM

Carcinoma of the Breast

Pathology—William C. MacCarty, Prof. Pathology, Mayo Foundation, Rochester, Minnesota.

Diagnosis and Differential Diagnosis—Dean Lewis, Prof. of Surgery, Johns Hopkins Hospital, Baltimore, Maryland.

Surgical Treatment—Arthur D. Bevan, Prof. of Surgery, Rush Medical College.

Radiological Treatment—Edward L. Jenkinson, Associate Porfessor of Radiology, Northwestern University.

Discussion: Richard H. Jaffe, Otto Saphir, Lewis L. McArthur, R. W. McNealy, George F. Thompson, Edwin M. Miller, Paul Oliver, Max Cutler, Pater A. Nelson, A. James Larkin.

Regular Meeting, Wednesday, October 17, 1934
LAY EDUCATIONAL PROGRAM—HEART DISEASE

The Principal Causes of Heart Disease....N. C. Gilbert
The Nature of the Increase of Heart Disease.....
.....Iago Gladston, New York, N. Y.

Regular Meeting, Wednesday, October 24, 1934
PROGRAM

"The Common Cold" (General Practitioner's Night)

"Influenza and the Common Cold"—Russell L. Cecil, Professor of Clinical Medicine, Cornell University, New York.

"Field Studies of the Common Cold"—Frank Jirka, Director, Department of Public Health State of Illinois.

"The Economic Cost of the Common Cold"—W. W. Bauer, Director, Bureau of Health and Public Instruction, American Medical Association.

"Treatment, Complications and Sequelae of the Common Cold"—Walter H. Nadler, Associate Professor of Medicine, Northwestern University Medical School.

Discussion: Robert Keeton, Milton Mandel, Anders Frick, Robert Black, Isadore Pilot, Francis Lederer, John Cavanaugh Harry L. Huber.

Demonstration prior to the meeting—X-ray Carcinoma of the Hands, Edward H. Ochsner.

RANDOLPH COUNTY

Chester, Illinois, Sept. 27, 1934.

The first meeting of the Randolph County Medical Society for the year 1934 and 1935 was held in the Illinois Security Hospital, Menard, Illinois, on the evening of September 27, 1934. The program consisted of, "The Relationship of Psychiatry in the Criminal Courts of Cook County." This was the main address of the evening and was given by Dr. Harry R. Hoffman, Director of the Behavior Clinic of Cook County, Chicago, Illinois. Other talks were given by Dr. James M. McManus, Managing Officer of the Illinois Security Hospital, who spoke on the relationship of his institution to the general practitioner. He invited all the medical societies in southern Illinois to make use of this institution for any clinical or medical material. The Honorable John Landesco, member of the Division of Pardons and Paroles, spoke of the relationship between parole work and psychiatric and medical factors. He stressed the importance of medical advice to the Parole Board. Medical and psychiatric cases were then presented by Dr. Hoffman, Dr. E. Ralph May, Institution Physician of the Illinois Security Hospital, and by Dr. David P. Philips, Psychiatrist of the Division of the Criminologist. The physicians who attended were taken through the wards where the cases were demonstrated.

The meeting was followed by an elaborate lunch prepared by the Institution Staff. Nine members of the Randolph County Medical Society were present and forty guests. The guests were from the following counties: Livingston, Macon, Perry, Jackson, Union, Randolph, Clinton, Cook and Peoria. Guests from St. Louis also attended. The purpose of having the initial meeting at the Illinois Security Hospital was twofold: First, it was an opportunity to receive information on psychiatric subjects, and, second, to acquaint the members of this county and adjoining counties of the purpose of the Illinois Security Hospital and the fact that its Staff and clinical material are available to all medical societies.

It is the plan of the Randolph Medical Society for the coming year to institute a postgraduate course for its members which will be conducted by speakers from out of the county. So far as possible authorities in his own field will be obtained as speakers. In this way it is hoped to give to the members of the society the latest information in the various branches of medicine. This first meeting included the relationship of psychiatry to courts, Parole Board and to the general practitioner.

E. Ralph May, M. D.,
Secretary, Randolph County Medical Society.

TRI-COUNTY MEDICAL SOCIETY

The 18th Annual Meeting of the Tri-County Medical Society, (Warren-Knox-Henry) was held at Monmouth on Thursday, October 18, 1934. This Society holds one annual meeting in rotation, at Monmouth, Galesburg, and Kewanee.

The first speaker on the program was Dr. Julius H. Hess, Professor of Pediatrics, University of Illinois College of Medicine, Chicago, and his subject was "The Present Status of Serum Therapy in Pediatrics." Dr. Hess reviewed the entire subject of the use of serums in the various infantile diseases, both artificial and natural serums being used. He told in detail of the use of blood transfusions in septicemias, the status of serums in scarlet fever, measles, infantile paralysis, pneumonia, and many other diseases. The paper was freely discussed by many of the guests at the meeting and many questions were asked which Dr. Hess answered thoroughly.

Dinner was served promptly at 6:30, and there were two dinner speakers on the program. Dr. Chas. S. Skaggs, President, Illinois State Medical Society, East St. Louis, and Dr. Gordon V. Harkness, President, Iowa State Medical Society, Davenport, Iowa, both gave highly interesting economic talks which were greatly appreciated by the large group present. The necessity of membership in their county medical society of all present day practitioners of medicine was stressed. Cooperation of the entire membership in their dealings with one another, and promoting contacts with various interested lay-groups, for the betterment of medical care, were also discussed in these interesting talks.

Following the dinner, Dr. Wm. R. Cubbins, Associate Professor of Surgery, Northwestern University Medical School, Chicago, gave an illustrated talk on "Fractures around the knee joint," this subject being illustrated by both motion and still pictures. Dr. Cubbins discussed in detail the many interesting common and unusual fractures of this nature encountered in practice giving the best methods of treatment of each type.

The third speaker on the program was Dr. Frederick H. Falls, Professor of Surgery, University of Illinois College of Medicine, whose subject was "Management of Eclamptogenic Toxemia." Dr. Falls discussed the toxemias occurring in pregnancy, and particularly stressed their early detection, and the proper methods of treatment. He reported a series of some five hundred consecutive cases encountered in his service, giving complete details, and a summary of the large group. This paper was thoroughly discussed by many members and guests in attendance.

The total attendance at the meeting was approximately 135, there being physicians present from 28 different counties. Visitors were present from Burlington, Davenport, Ottumwa, Keokuk and Fort Madison, Iowa. Quincy, Peoria, Rock Island, Moline, Aledo, Macomb, Canton, Rushville, practically every city and town in the Tri-counties (Warren, Knox, and Henry) and from many other smaller towns in Western, and West Central Illinois. Although the membership in the Tri-County Society is limited to the members of the county medical societies of three counties, a mailing list of five hundred physicians is maintained, all of whom are each year extended an invitation to the meetings. All present at the meeting were unanimous in the belief that meetings conducted by several county medical societies jointly, are desirable, and should be continued.

Charles P. Blair, M. D.,
Secretary, Warren County Medical Society.

Marriages

Thomas Joseph Conley Jr., Chicago, to Miss Elizabeth M. Cardwell of Evanston, October 10.

Claire M. Dixon, Ina, Ill., to Miss Lucile Montane of Sacramento, Calif., in East St. Louis, Ill., September 8.

Michael Gleason, Mendota, Ill., to Miss Celeste Brahaney of Buffalo, September 4.

John Leonard Probasco, Bloomington, Ill., to Miss Laurastine Welch of Lexington, August 18.

Cyril Fantus Sherman, Danville, Ill., to Miss Jennie Eylene Worden of Hillsboro, Wis., May 27.

Personals

Dr. Frank F. Maple, Chicago, addressed the Fulton County Medical Society, September 27, on "Septic Abortion."

Dr. Gilbert FitzPatrick, Chicago, discussed cancer before the Marion County Medical Society, September 27.

Dr. Robert von der Heydt discussed "The Ageing Lens" before the Chicago Ophthalmological Society, October 15.

Dr. Aaron Arkin, Chicago, addressed the Rock Island County Medical Society in Moline, September 11, on lesions of the gastro-intestinal tract.

Dr. Cyrus C. Sturgis, Ann Arbor, addressed the Peoria City Medical Society, October 2, on "Treatment of Secondary Anemias."

Dr. Clarence O. Sappington, Chicago, addressed the Peoria City Medical Society, October 16, on occupational diseases.

Dr. Leon Unger addressed the Chicago Society of Allergy, among others, October 15, on "Turpentine Abscesses in Treatment of Intractable Asthma."

At a meeting of the Chicago Orthopedic Club, October 12, Dr. Arthur Steindler, Iowa City, among others, discussed "Tuberculosis of the Wrist."

Dr. William R. Cubbins, Chicago, addressed the Rock Island County Medical Society, October 9, on "Fractures of the Lateral Condyle of the Tibia."

At a meeting of the Will-Grundy County Medical Society, October 10, Dr. Philip Lewin presented a paper on "The Classification, Etiology and Treatment of Arthritis."

Dr. James H. Hutton, Chicago, addressed the DeWitt County Medical Society, October 4, on the endocrine aspects of essential hypertension.

Dr. Max Thorek addressed the New York Polyclinic Medical School and Hospital on "A New Method of Obliterating the Gallbladder by Electrosurgical Means" on October 1, 1934.

Dr. Oscar B. Nugent lectured to the Evening Auxiliary Austin Women's Club at the Austin Baptist Church on "The Eyes of India," Oct. 22. Dr. Nugent was also invited to show his travelogue on India to the Medinah Medical Staff and Officers on Oct. 24 at the Hamilton Club. He addressed the Central Y. M. C. A. Evening Class Oct. 26, on the subject "Cataract."

Dr. Paul R. Cannon, professor of pathology University of Chicago, presented the sixth annual William T. Belfield Lecture at the Palmer House, October 25, under the auspices of the Chicago Urological Society. His subject "Bacterial Localization."

Dr. Traian Leucutia, Detroit, discussed "Further Contributions to the Problems of Superhigh Voltage Roentgen Therapy" before the Chicago Roentgen Society, October 11, and Dr. Benjamin H. Orndoff gave a report on the fourth International Congress of Radiology.

Dr. C. R. G. Forrester gave a talk before the Tenth District Medical Society of Indiana, in Gary, October 31, on the subject "Reduction of Fractures Under Local Anesthesia with Ambulatory Treatment."

Dr. Sidney A. Portis addressed the Porter County Medical Society, Valparaiso, Indiana, September 25 on the "Medical Aspects of Gall Bladder Disease."

Dr. Geza deTakats addresssed medical societies in Vienna and Budapest, last month.

News Notes

—At a meeting of the Will-Grundy County Medical society at Joliet, September 26, Dr. Francis L. Lederer, Chicago, spoke on "Cancer About the Head and Neck—A Critical Analysis of the Available Therapeutic Measures."

—Speakers before the Chicago Pediatric Society, October 16, were Drs. Arthur H. Parmelee on "Physiologic Changes of Adolescence" and Bert I. Beverly on "Psychologic Problems of the Adolescent."

—A symposium on collapse therapy was presented before the Chicago Tuberculosis Society, October 11, by Drs. Frederick Tice, Allan J. Hruby, Karl J. Henrichsen, Robert F. Berry and Henry C. Sweany.

—The fourth annual Mayo Lecture in Surgery was delivered by Sir Harold Gillies, London, at Thorne Hall, McKinlock Campus, Northwestern University School of Medicine, October 23, on "The Development and Scope of Plastic Surgery."

—At a meeting of the Chicago Gynecological Society, October 19, the speakers were Drs. Fred L. Adair and M. Edward Davis on "Chronic Atrophic Dermatitis of the Vulva," and Carl Henry Davis, Milwaukee, "Use of the Colposcope in the Diagnosis of Cervical Lesions."

—Speakers before the Adams County Medical Society in Quincy, October 8, were Drs. Dan G. Stine, Columbia, Mo., on "Value of the Leuko-

cyte Count in Pulmonary Tuberculosis," and Marcus Pinson Neal, Columbia, Mo., "The Leukocyte Blood Pictures in Acute Infections."

—At a special meeting of the Will-Grundy County Medical Society in Joliet October 3, Drs. Joseph Colt Bloodgood, Baltimore, and Max Cutler, Chicago, were the speakers, on cancer. Dr. Philip Lewin, Chicago, addressed the society, October 10, on "Classification Etiology and Treatment of Arthritis."

—At a meeting of the Chicago Pathological Society October 8, Dr. Edwin F. Hirsch, among others, sopke on "Generalized Osteosclerosis with Chronic Polycythemia Vera." The presidential address was given by Dr. Isadore Pilot on "Hemolytic Streptococci, Their Present Status and Relationship to Certain Clinical Entities."

—Dr. Arthur Steindler, Iowa City, addressed the Chicago Orthopedic Club, October 12, on "Tuberculosis of the Wrist." Other speakers included Drs. Frank G. Murphy on "Traumatic Rupture of Quadriceps Tendon Above the Patella with Surgical Repair" (case presentation), and Edson B. Fowler, Evanston, Ill., "Internal Fixation of Fractures with Horn Cortico-Medullary Technic."

—Dr. James P. Leake, senior surgeon, U. S. Public Health Service, Washington, D. C., gave an illustrated lecture before a joint meeting of the Institute of Medicine of Chicago and the Chicago Society of Internal Medicine at the Chicago Woman's Club, October 26. His subject will be "Poliomyelitis, with Special Reference to Epidemiology." Dr. Leake has been in California for several months studying the epidemic there.

—The management of children's behavior problems is the theme of a series of lectures to be given at the Institute for Juvenile Research, 907 South Lincoln Street, under the auspices of the University of Illinois College of Medicine. Lectures will be delivered on Tuesdays, October 9 to November 27. For a limited number of physicians these lectures may be supplemented by clinical experience in the treatment of children's behavior problems under supervision. Dr. Paul L. Schroeder is director of the Institute of Juvenile Research.

—Medical practice and government plans for economic security were discussed at a meeting of the North Side Branch of the Chicago Medical Society in Thorne Hall, McKinlock Campus, Northwestern University School of Medicine, October 8, as follows:

Dr. Olin West, Secretary and General Manager, American Medical Association, "The Situation Today."

Dr. Rosco G. Leland, Director, bureau of medical economics, American Medical Association, "What Medicine Is Doing."

Dr. Morris Fishbein, Editor, THE JOURNAL of the American Medical Association, "What Medicine Should Do."

Frederick A. Britten, congressman, ninth district, "The Legislator's Viewpoint."

—A series of graduate lectures on "The Physiological Basis of Individuality in the Early Embryo" were delivered at the University of Illinois College of Medicine, October 15-20, by Arthur R. Moore, Ph.D., professor of general physiology, University of Oregon, Eugene. Dr. Moore presented the following lectures on the dates indicated:

The Cytoplasm as a Biophysical System, October 15.

Special Cytoplasmic Structures, October 16.

The Individual in Motor Reactions of Simple Forms, October 17.

Phylogenetic Beginnings of the Central Nervous System, October 18.

Modifiability of Reactions, October 18.

Lectures were given in the Hall of Science at the World's Fair during October by the following physicians. A committee of the Chicago Medical Society arranged the program through the Educational Committee of the Illinois State Medical Society:

Monday, October 1—"How to Get the Best of Asthma," William L. Beecher, M. D.

Tuesday, October 2—"Infections of the Skin," Harry M. Hedge, M. D.

Wednesday, October 3—"Cancer," Gilbert Fitzpatrick, M. D.

Thursday, October 4—"Accidents Due to 20th Century Speed," John R. Harger, M. D.

Friday, October 5—"Gall Bladder Disease," I. E. Bishkow, M. D.

Saturday, October 6—"Periodic Health Examinations," R. K. Packard, M. D.

Monday, October 8—"Significance of Bloody Urine," Davis H. Pardoll, M. D.

Tuesday, October 9—"Fractures and Other Injuries," M. H. Hobart, M. D.

Wednesday, October 10—"The Dangerous Forties," Theodore Bacmeister, M. D.
Thursday, October 11—"Problems of Deafness," Francis L. Lederer, M. D.
Friday, October 12—"Cancer," J. D. Willems, M. D.
Saturday, October 13—"Crippled Children," Charles M. Pease, M. D.
Monday, October 15—"Some of the Contagious Diseases and Their Prevention," Proctor Cook Waldo, M. D.
Tuesday, October 16—"Stammering or Stuttering," Elmer L. Kenyon, M. D.
Wednesday, October 17—"The Personality of the Growing Child," Paul L. Schroeder, M. D.
Thursday, October 18—"Infantile Paralysis," Philip Lewin, M. D.
Friday, October 19—"Lecture and Demonstration of Apparatus—Occupational Diseases," C. O. Sappington, M. D.
Saturday, October 20—"Cancer of the Skin," B. Barker Beeson, M. D.
Monday, October 22—"Cancer," Frank L. Rector, M.D.
Tuesday, October 23—"Heart Disease," C. C. Maher, M. D.
Wednesday, October 24—"Feet and Posture," Frederick C. Test, M. D.
Thursday, October 25—"The Story of Blood Transfusion," R. W. McPherron, M. D.
Friday, October 26—"Cancer," Henry Schmitz, M. D.
Saturday, October 27—"Is It Safe to Have a Baby?" Charles Edwin Galloway, M. D.
Monday, October 29—"What Can Be Done for Sinus Disease?" George E. Shambaugh, Jr., M. D.
Tuesday, October 30—"The Crippled Child Ten Years from Now," Harold A. Sofield, M. D.
Wednesday, October 31—"The Human Eye and Its Care," C. G. Darling, M. D.

Committee—Dr. Wilbur Post, Dr. Julius H. Hess, Dr. Hugh N. MacKechnie, Chairman.

Deaths

George Enos Burdick, Hines, Ill.; University of the City of New York Medical Department, 1888; served during the World War; on the staff of the Veterans' Administration Facility; aged 69; died suddenly, September 24, in Glen Ellyn, of coronary occlusion.

Oral De Witt Cunningham, Rockford, Ill.; State University of Iowa College of Medicine, Iowa City, 1924; a Fellow, A. M. A.; aged 36; died, September 10, of agranulocytic angina.

Fred Danziger, Chicago; University of Illinois College of Medicine, Chicago, 1929; aged 33; died, October 12, of carcinoma of the rectum.

Frank Eyre, North Henderson, Ill.; University of Pennsylvania School of Medicine, Philadelphia, 1876; aged 80; died, August 18, of heat exhaustion and myocarditis.

Douglas Nebraska Harris, Prentice, Ill.; Northwestern Medical College, St. Joseph, 1887; member of the Illinois State Medical Society; aged 80; died, September 8, of uremia.

Clarence Hugh Harwood, Charleston, Ill.; University of Louisville (Ky.) School of Medicine, 1910; a Fellow, A. M. A.; formerly mayor; medical director and part owner of the Oakwood Hospital; aged 51; was killed, September 1, in an automobile accident.

James Arthur Kirk, Danville, Ill.; Hospital College of Medicine, Louisville, Ky., 1904; served during the World War; aged 53; on the staff of the Veterans' Administration Facility, where he died, September 3, of Hodgkin's disease and chronic pulmonary tuberculosis.

Julius H. Krueger, Chicago; Hahnemann Medical College and Hospital, Chicago, 1887; aged 88; died, September 25, of cerebral hemorrhage and arteriosclerosis.

Constantine S. Krysinski, Chicago; Hering Medical College, Chicago, 1909; for many years on the staff of St. Elizabeth's Hospital; aged 47; died, August 1, of carcinoma of the lung.

Russel Bennett Miller, Chicago; Columbus Medical College, 1880; aged 77; died, September 19, at Gleason, Wis., of pulmonary tuberculosis.

Samuel Sal Murnick, Chicago; Loyola University School of Medicine, Chicago, 1922; aged 40; died, September 16,, of organic heart disease.

Frank John Novak, Sr., Chicago; Rush Medical College, Chicago, 1885; a Fellow, A. M. A.; on the staff of the Frances E. Willard Hospital; aged 70; died, October 3, at his home in Riverside, Ill., of coronary thrombosis.

Frank Joseph Patera, Chicago; Rush Medical College, Chicago, 1883; a Fellow, A. M. A.; on the staff of St. Mary of Nazareth Hospital; aged 73; died, September 12, at Rhinelander, Wis., of hypertrophy of the prostate gland and chronic nephritis.

Arthur F. Schellschmidt, Red Bud, Ill.; Hospital College of Medicine, Louisville, 1892; aged 64; on the staff of St. Clement's Hospital, where he died, September 17, of pneumonia.

Clarence Earl Smart, Granville, Ill.; Chicago College of Medicine and Surgery, 1917; member of the Illinois State Medical Society; served during the World War; on the staff of St. Margaret's Hospital, Spring Valley; aged 45; died suddenly, August 30, of cerebral hemorrhage.

Thomas T. Waggoner, Freeport, Ill.; Barnes Medical College, St. Louis, 1899; member of the Illinois State Medical Society; on the staff of St. Francis Hospital; aged 71; died, September 2, of heart disease.

Timothy Charles Weber, West Salem, Ill.; Barnes Medical College, St. Louis, 1897; served during the World War; aged 64; died, August 28, of tuberculosis.

John Harrison Wedig, Wood River, Ill.; Chicago College of Medicine and Surgery, 1910; a Fellow, A. M. A.; served during the World War; aged 49; died, September 7, in St. Joseph's Hospital, Alton, of sarcoma of the lung.

Jeremiah Calfer Wilson, Greenville, Ill.; St. Louis College of Physicians and Surgeons, 1883; aged 74; died, September 9, of heart disease.

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